UNITED STATES ENVIRONMENTAL PROTECTION AGENCY

Comments of Safer Chemicals Healthy Families et al. on Risk Evaluation Problem Formulation Documents for Ten Chemical Substances under the Toxic Substances Control Act

Submitted via Regulations.gov (August 16, 2018)

1,4-Dioxane. Docket ID No.: EPA-HQ-OPPT-2016-0723.

1-Bromopropane. Docket ID No.: EPA-HQ-OPPT-2016-0741.

Asbestos. Docket ID No.: EPA-HQ-OPPT-2016-0736.

Carbon Tetrachloride. Docket ID No.: EPA-HQ-OPPT-2016-0733.

Cyclic Aliphatic Bromide Cluster (Hexabromocyclododecane or HBCD). Docket ID No.: EPA-HQ-OPPT-2016-0735.

Methylene Chloride. Docket ID No.: EPA-HQ-OPPT-2016-0742.

N-Methylpyrrolidone (NMP). Docket ID No.: EPA-HQ-OPPT-2016-0743.

Pigment Violet 29 (Anthra[2,1,9-def:6,5,10-d'e'f]diisoquinoline-1,3,8,10(2H,9H)-tetrone). Docket ID No.: EPA-HQ-OPPT-2016-0725.

Trichloroethylene (TCE). Docket ID No.: EPA-HQ-OPPT-2016-0737.

Tetrachloroethylene (also known as Perchloroethylene). Docket ID No.: EPA-HQ-OPPT-2016-0732.

Safer Chemicals Healthy Families (SCHF) and the undersigned groups submit these comments on the problem formulations developed by the Environmental Protection Agency (EPA) on the initial 10 chemicals selected for risk evaluations under the newly enacted Frank R. Lautenberg Chemical Safety for the 21st Century Act (LCSA).[1]

SCHF leads a coalition of national and grassroots organizations committed to assuring the safety of chemicals used in our homes, workplaces and the many products to which our families and children are exposed each day. SCHF and its partners took a leadership role during the LCSA legislative process, advocating the most protective and effective legislation possible to reduce the risks of toxic chemicals in use today.

These comments address crosscutting legal and policy issues common to the 10 chemicals as well as several chemical-specific issues. We are submitting our comments to all ten of the EPA dockets. The comments

[1] 83 Federal Register 26998 (June 11, 2018).

build on earlier SCHF submissions, including our September 19, 2017 comments on the EPA scoping documents on the 10 chemicals. Many SCHF partner organizations are also commenting on the problem formulations and we support these comments.

Organizations joining these comments are:

Alaska Community Action on Toxics
Alliance of Nurses for Healthy Environments
Asbestos Disease Awareness Organization
Center for Environmental Health
Clean and Healthy New York
Clean Production Action
Clean Water Action (National)
Clean Water Action (Connecticut)
Colorado PIRG (CoPIRG)
Earthjustice
Environmental Health Strategy Center
Healthy Building Network
League of Conservation Voters
Learning Disabilities Association of America
Maryland PIRG
Natural Resources Defense Council
Science and Environmental Health Network
Texas PIRG (TexPIRG)
Toxic-Free Future
U.S. PIRG
United Steelworkers
WashPIRG
WE ACT for Environmental Justice
Women for a Healthy Environment

OVERVIEW

Through LCSA, Congress amended the Toxic Substances Control Act (TSCA) to establish a new framework for conducting timely, comprehensive and science-based risk evaluations for chemicals of concern. The law provides that EPA's evaluations must be strictly risk-based and must result in a definitive determination of whether the evaluated substance as a whole presents an unreasonable risk of injury to health and the environment across its life cycle, without regard to cost and other non-risk factors. In conducting risk evaluations, EPA must address risks not only to the general population but also to "potentially exposed or susceptible subpopulations," including the elderly, children, pregnant women and workers.

On December 19, 2016,[2] as required by section 6(b)(2)(A) of TSCA, EPA selected 10 chemicals for initial risk evaluations. These precedent-setting evaluations address substances with widespread exposure and known health hazards. How EPA evaluates the risks of these chemicals will be critical to whether the public and policymakers are fully informed about the threats they pose to health and the environment. This in turn will determine whether EPA follows through with effective risk reduction measures under section 6(a) of TSCA that protect at-risk populations. The initial evaluations will also lay the groundwork for overall TSCA implementation and thus determine whether EPA establishes the robust and protective chemical risk management program that LCSA calls for.

Unfortunately, the 2017 scoping documents and more recent problem formulations make it increasingly apparent that the initial 10 evaluations will fall far short of the expectations of Congress and the requirements of the law. Through a combination of questionable exclusions and loopholes, failure to require necessary testing, deviations from accepted scientific methods and refusal to accept previous peer reviewed determinations of risk, the Agency is on a path to produce evaluations that ignore important exposure

[2] 81 Federal Register 91927

pathways and at-risk populations, disregard evidence of adverse effects and reach misleading and incomplete conclusions that understate risks and weaken public health protection.

The many shortcomings of the scoping documents and problem formulations are compounded by the June 11 TSCA document for applying "systematic review" methods in the TSCA risk evaluations. As explained in our separate comments on this document, it would require data on the 10 chemicals to be reviewed using an arbitrary set of numerical criteria for study quality that has not been peer reviewed and is in conflict with other systematic review approaches used within EPA and by other federal agencies that have been endorsed by authoritative bodies like the National Academy of Sciences (NAS). Application of the TSCA systematic review document will unjustifiably restrict the body of evidence that informs EPA judgments about risk and hamper the Agency's ability to use the most relevant and meaningful data for decision-making on the 10 chemicals.

Because the 10 risk evaluations are likely to deviate dramatically from the goals of the law and take a large step backward in protecting public health, EPA should put them on hold, rethink how they are being conducted, and reinitiate them in accordance with the law and principles of sound science.

SUMMARY OF KEY POINTS

As described more fully in the body of these comments, we have the following fundamental concerns about the approach to risk evaluation reflected in EPA's scoping documents and problem formulations:

- Congress intended the scope of risk evaluations to be defined within six months after their initiation. Problem formulations are not an authorized step in the risk evaluation process and cannot be used to revisit issues of scope after the Agency has issued a scoping document in accordance with section 6(b)(4)(D). The problem formulations on the 10 chemicals are unlawful under TSCA because they go far beyond the scoping documents in excluding uses, exposures and hazards from the risk evaluations. (Section I, page 6)
- In direct contrast to the scoping documents, all the problem formulations provide that EPA will not consider environmental exposure pathways that could be addressed under other laws administered by EPA. This approach would remove all environmental exposure pathways – a significant contributor to human health risk for many chemicals – from the TSCA risk evaluation process. This dramatic narrowing of TSCA's scope is contrary to the plain language of the law and will defeat the central purpose of TSCA reform – to conduct comprehensive risk evaluations on ubiquitous chemicals that examine the impacts on health and the environment of all of the diverse pathways and modes of release that may result in harm. (Section II, pages 7-12)
- In an extension of this approach, several of the problem formulations indicate that EPA will not evaluate the risks of general population exposure to the 10 chemicals. However, if the presence of a chemical in environmental media – and therefore exposure to the chemical by the general population – is attributable to its "conditions of use," there is no basis for excluding this background level of exposure from EPA's risk evaluation. Moreover, EPA cannot perform its obligation under the law to "integrate and assess" information on exposure if it ignores the contribution of general population exposure to the

overall risk that a chemical poses to subpopulations that have additional sources of exposure. (Section III, pages 12-13)

- More broadly, neither the scoping documents nor the problem formulations shed any light on how EPA risk evaluations will account for multiple pathways of exposure by the general population or subpopulations. Instead, it appears that EPA will examine each source of exposure in isolation and will not consider either the combined effect of multiple exposures or the contribution of environmental releases to overall exposure and risk. This is a violation of TSCA. (Section IV, pages 13-14)
- Despite the deep concerns of commenters, the problem formulations reaffirm EPA's exclusion from its risk evaluations of ongoing use and disposal of chemical products that are no longer being manufactured (so-called "legacy uses"). This use and disposal clearly falls within the TSCA definition of "conditions of use" and its exclusion violates the plain language of the law. As the case of asbestos illustrates, discontinued products may be ubiquitous in the built environment and their contribution to current and future exposure and risk may greatly dwarf that of the few products that remain in commerce. To ignore this source of risk would deprive the public, scientists and regulators of important information about threats to public health and prevent policymakers from taking meaningful action to protect at-risk populations. (Section V, pages 14-16)
- Further narrowing the scope of risk evaluations, EPA has determined that it will not address recently discontinued uses of chemicals. The goals of TSCA would be defeated if manufacturers of unsafe chemicals could circumvent scrutiny simply by ceasing production for specific uses before EPA completes a risk evaluation of those uses and then later re-entering the marketplace free from any restriction or determination of risk. This scenario is of particular concern where the product phase-out is in response to agency scrutiny and intended to avoid the consequences of an adverse risk finding and subsequent regulatory action. Although EPA claims that discontinued uses are not "conditions of use" as defined in TSCA, the future resumption of these uses can be "reasonably foreseen" and thus would satisfy the statutory definition. By including such uses in its risk evaluation, EPA could then ban or restrict them permanently under section 6(a), providing certainty to the marketplace and long-term public health protection. (Section VI, pages 16-18)
- Our groups have repeatedly called for EPA to identify data gaps that limit its ability to reach definitive conclusions about the health and environmental effects of the 10 chemicals. However, the problem formulations make a minimal effort to identify the absence of data on the 10 chemicals and address how lack of information will impact the conclusions reached in the risk evaluations. In the face of material data gaps, an unqualified conclusion that a chemical does not "present an unreasonable risk of injury" to health could not be defended under TSCA and would misinform the public about the chemical's safety. Thus, EPA should be explicit about the health and environmental end-points that lack adequate data and exclude these end-points from its determinations of unreasonable risk. It should also use its TSCA authorities to require manufacturers to conduct testing to develop adequate data for a defensible risk evaluation so that future assessments can be informed by a comprehensive dataset. (Section VII, pages 18-23)
- The problem formulations indicate that conditions of use that present *de minimis* risks will not be further analyzed or addressed in risk evaluations. However, EPA has provided no general criteria for

determining levels of exposure that are insignificant. Nor has it provided any information to demonstrate that the uses it plans to drop lack meaningful exposure potential, either in themselves or in relation to their contribution to overall exposure. EPA may have some latitude to devote greater effort to some exposure scenarios than others, but this does not excuse ignoring particular conditions of use based on the unsubstantiated claim that their risks are negligible. (Section VIII, pages 23-24)

- As the asbestos risk evaluation illustrates, EPA has also dropped from consideration significant health end-points known to be linked to exposure to the chemical. This omission is likewise contrary to TSCA's comprehensive approach to evaluating risk. (Section IX, pages 24-25)
- Six of the 10 chemicals – asbestos (and Libby amphibole asbestos), trichloroethylene (TCE), methylene chloride (MC), carbon tetrachloride (CTA), perchloroethylene (PERC) and 1,4-dioxane – have been assessed under the EPA Integrated Risk Information System (IRIS). The IRIS process is the Agency's authoritative mechanism for reviewing available studies and characterizing the health effects of chemicals. The problem formulations, however, indicate that EPA will revisit the interpretation of studies already evaluated in IRIS using its highly questionable TSCA "systematic review" method that has not been peer reviewed. This may lead to departures from IRIS determinations of the "best available science" and "weight of the evidence." Reopening IRIS findings would harm the public by prolonging uncertainty on issues that have been addressed and resolved through an authoritative and transparent process. In rare cases where significant new data (since the IRIS assessment) are available, the EPA TSCA program should rely on the IRIS program to review, assess, and if appropriate incorporate any new information using a systematic review method that is consistent with the state of the science. (Section X, pages 25-28)
- EPA has proposed to ban certain uses of TCE and N-methylpyrrolidone (NMP) under TSCA section 6(a) based on comprehensive exposure and risk assessments of these uses, including its peer reviewed IRIS assessments on TCE. However, the problem formulations indicate that EPA intends to reopen these completed assessments and delay regulatory action despite serious threats to public health. This is unjustified and unnecessary. EPA should finalize the proposed rules without delay. (Section XI, pages 28-29)
- Occupational exposure is significant for nearly all of the 10 chemicals and should be a major focus of EPA's risk evaluations. The problem formulations indicate that when evaluating occupational risks, the Agency will heavily weigh applicable workplace standards. Although these standards may be relevant, EPA should not presume that they are fully protective of workers or that their existence can be equated with the absence of unreasonable risk. OSHA and EPA apply differing standards of protection by law; several OSHA standards are obsolete and do not reflect best available science; OSHA standards do not cover all workers with exposure to regulated chemicals; compliance with OSHA standards is uneven and variable; and as EPA has recognized, some of the industrial hygiene strategies embodied in OSHA standards – such as labels and respirators – are known to be of limited effectiveness in protecting workers. EPA should explicitly recognize these considerations in determining whether risks to workers are unreasonable under TSCA. (Section XII, pages 29-32)

I. The Problem Formulations Have No Basis in the Law and Improperly Narrow the Scope of the 10 Risk Evaluations

Section 6(b)(4)(D) of amended TSCA provides that, "not later than 6 months after the initiation of a risk evaluation," EPA must "publish the scope of the risk evaluation to be conducted, including the hazards, exposures, conditions of use and the potentially exposed or susceptible subpopulations the Administrator expects to consider." There is no authorization in the law for issuing a "problem formulation" document at a later point in time to further refine, expand or narrow the scope of the risk evaluation. Nor is this step identified in EPA's final risk evaluation framework rule issued under TSCA section 6(b)(4)(B).

Nonetheless, when it released its scoping documents for the 10 chemicals in June 2017, EPA announced that it was also developing problem formulations.[3] It justified this step on the basis that it had been unable to process all the information gathered during the scoping process and the scoping documents were not as "refined or specific" as EPA had hoped. Although the problem formulations may have performed a useful role under these unique circumstances, we do not support repeating this step for additional risk evaluations that EPA conducts. The intent of Congress was to provide clear notice to the public of the scope of risk evaluations within six months after they are initiated. This goal will be undermined if EPA retains the discretion to revisit issues of scope throughout the risk evaluation process and to continuously modify the hazards, uses and exposures that its evaluations will address.[4] Thus, problem formulation should be a one-time activity, limited to the special case of the first 10 chemicals, and not part of the risk evaluation process in the future.

We are also concerned that the problem formulations on the 10 chemicals go far beyond the scoping documents in excluding uses, exposures and hazards from the risk evaluations. Not only are these exclusions not justified under TSCA[5] but they narrow the evaluation significantly after its scope had been established in accordance with section 6(b)(4)(D). Since problem formulation is not a recognized step in the risk evaluation process or a substitute for scoping under LCSA, it cannot be used to narrow a risk evaluation's scope after-the-fact. Thus, the additional exclusions established in the problem formulations are unlawful.

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[3] 82 Fed. Reg. 31,592 (July 7, 2017).

[4] Thus, instead of taking comments on proposed scoping documents and addressing them in final scoping documents issued six months after a risk evaluation is initiated, EPA is now requesting comments on scope issues 20 months into the risk evaluation process. EPA plans to release draft risk evaluations by the end of 2018. Thus, it will be unable to review the comments and modify the evaluations without delaying their completion. In practice, this creates a high likelihood that the comments will be ignored. EPA admits as much by acknowledging that it plans to respond to the comments only when the risk evaluations are final.

[5] EPA's final risk evaluation rule, in contrast to its proposal, would permit the Agency to select which conditions of use to address in risk evaluations. 82 Fed. Reg. 33726 (July 20, 2017). SCHF and several of its partner organizations argued in their comments on the proposal that the law requires the Agency to address all conditions of use in its evaluations. Along with several other groups, SCHF is challenging EPA's contrary interpretation in its petition for judicial review of the risk evaluation rule. *Safer Chemicals Healthy Families v. EPA*, 17-72260 (9th Cir.) Regardless of the outcome of this challenge, we believe that EPA has no basis to narrow the risk evaluation to exclude conditions of use once they have been included in its scope.

II. EPA's Extreme Approach of Removing All Environmental Exposure Pathways from Risk Evaluations Is Contrary to the Plain Language and Structure of TSCA and Will Defeat the Central Purpose of TSCA Reform

In direct contrast to the scoping documents, all 10 of the problem formulations provide that EPA will not evaluate the risks of "exposure pathways that are under the jurisdiction of regulatory programs and associated analytical processes carried out under other EPA-administered environmental statutes – namely, the Clean Air Act (CAA), the Safe Drinking Water Act (SDWA), the Clean Water Act (CWA), and the Resource Conservation and Recovery Act (RCRA)."[6] EPA's rationale for this blanket exclusion is that it "believes that certain programs under other Federal environmental laws adequately assess and effectively manage the risks for the covered exposure pathways." As the Agency explains, "[t]he provisions of various EPA-administered environmental statutes and their implementing regulations represent the judgment of Congress and the Administrator, respectively, as to the degree of health and environmental risk reduction that is sufficient under the various environmental statutes."

Since the laws cited by EPA potentially apply to all releases into the environment, the effect of EPA's approach would be to remove environmental exposure pathways in their entirety from the TSCA risk evaluation process. This extreme approach is without any basis in the text of the law and will defeat the central purpose of TSCA reform – to conduct comprehensive risk evaluations on ubiquitous chemicals that examine the impacts on health and the environment of all of the diverse pathways and modes of release that may result in harm. Environmental media – air, surface water, groundwater, drinking water and waste – are known and pervasive sources of exposure for many substances. Any risk evaluation that fails to account for their contribution to total exposure will provide the public with a misleading and incomplete account of their potential to harm human health and fail to identify critical opportunities for risk reduction.

A. TSCA Risk Evaluations Must Examine Total Risk and Consider All Contributors to Exposure and Conditions of Use

Risk evaluations under TSCA section 6(b)(4)(A) must determine "whether a chemical substance presents an unreasonable risk of injury to health or the environment." These evaluations must therefore examine the totality of risks presented by the substance, taking into account all contributors to exposure, including not just its presence in the workplace or consumer products but its releases into the environment. Indeed, under the plain language of the statute, EPA's focus expressly includes risks to the *environment* in addition to human health. "Environment" is defined in section 3(6) to include "air, water and land and the interrelationship which exists among and between air, water and land and all living things." If EPA excludes the chemical's presence in environmental media (air, water and soil) and the impacts on the environment of that presence on humans and other living things, then it cannot meet its obligation to determine environmental risks.

Section 6(b)(4)(A) also provides that a risk evaluation must also determine the substance's risks under "the conditions of use." This broad term spans the entire life cycle of a chemical. It is defined under

[6] See, e.g., Problem Formulation of the Risk Evaluation for Carbon Tetrachloride (May 2018) at 13.

section 3(4) to mean "the circumstances . . . under which a chemical substance is intended, known or reasonably foreseen to be manufactured, processed, distributed in commerce, used or disposed of." The "circumstances" to which the definition applies clearly include air emissions and water discharges from industrial facilities as well as releases to environmental media during disposal. For EPA to exclude all such environmental releases from its risk evaluations would remove from the application of the law a large category of "conditions of use" that Congress directed EPA to address.[7]

B. Environmental Exposure Pathways Are Central to Chemical Prioritization, Risk Evaluation and Regulation under Section 6 of TSCA

Other provisions in section 6 confirm the need to consider environmental releases as part of chemical prioritization and risk evaluation. For example, storage near significant sources of drinking water is a factor that EPA must examine in its process for designating chemicals as high- or low-priority under section 6(b)(1)(A). Similarly, under both this provision and section 6(b)(2)(D), chemicals with significant potential for persistence, bioaccumulation and toxicity (PBTs) must receive preference in the selection of substances for high-priority listing. PBTs are of concern because of their presence in environmental media and potential to concentrate in animals and humans as they are distributed in air, water and soil taken up the food chain. If EPA does not consider environmental release pathways of PBTs in evaluating their risks, it would be pointless to designate them as high-priority since the ensuing evaluation could not meaningfully address the contribution of environmental exposure pathways to total risk.

Paralleling the expansive definition of "conditions of use," the regulatory authorities in section 6(a) of the law empower EPA to take a broad array of actions to restrict chemical exposures and releases in order to eliminate unreasonable risks to health and the environment. Under the original law, EPA in fact used section 6(a) on a number of occasions to curtail environmental releases of toxic chemicals.[8] Indeed, section 6(a)(6)(A) authorizes EPA to impose a "requirement prohibiting or otherwise regulating any manner or method of disposal of such substance or mixture, or of any article containing such substance or mixture, by its manufacturer or processor or by any other person who uses, or disposes of, it for commercial purposes." The authority to regulate disposal (a broad concept that can include virtually any release of wastes into air, water or land) would be meaningless if EPA did not use risk evaluations under section 6(b) to identify disposal activities that present an unreasonable risk of injury and are subject to restriction under section 6(a).

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[7] As SCHF and its co-petitioners have argued in their brief in *Safer Chemicals Healthy Families v. EPA,* the statute gives EPA no discretion to exclude any conditions of use from risk evaluations, let alone the broad universe of environmental releases that occur during manufacture, processing, use, distribution in commerce and disposal of a chemical substance.

[8] Of the 6 existing chemicals EPA regulated under section 6 under the original law, the prevention of environmental releases was the basis for three of these regulatory actions. In 1978, EPA banned nonessential uses of fully halogenated chlorofluoroalkanes as propellants in aerosol spray containers because of concerns that these chemicals were destroying the upper atmosphere's ozone layer. In 1980, EPA promulgated a rule prohibiting Vertac Chemical Company and others from removing for disposal certain wastes containing 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) stored at Vertac's Jacksonville, Arkansas, facility. The rule also required any persons planning to dispose of TCCD contaminated wastes to notify EPA 60 days before their intended disposal. In 1994, EPA promulgated a rule to eliminate emissions of hexavalent chromium from comfort cooling towers.

C. TSCA Legislative History Demonstrates that the Law Was Intended to Address Environmental Releases that May Be Within the Purview of Other Laws

If Congress had intended a blanket exemption for environmental releases from risk evaluations under section 6(b) and regulation under section 6(a), it surely would have said so explicitly given the far-reaching impact of such an exemption. Not only is there no such exemption in the law, but its legislative history and structure demonstrate that Congress intended TSCA to provide a comprehensive framework for identifying and managing chemical risks, including those that derive from environmental exposure pathways and could be addressed under other environmental laws.

The comprehensive scope of TSCA was underscored in the legislative history of the original law. Congress recognized that then-existing environmental laws were "clearly inadequate" to address the "serious risks of harm" to public health from toxic chemicals. H.R. Rep. No. 94-1341, at 7 (1976); see S. Rep. No. 94-698, at 3 ("[W]e have become literally surrounded by a manmade chemical environment. ... [T]oo frequently, we have discovered that certain of these chemicals present lethal health and environmental dangers."). While other federal environmental laws focused on specific media, such as air or water, none gave EPA authority to "look comprehensively" at the hazards of a chemical "in total." S. Rep. No. 94-698, at 2. Congress designed TSCA to fill these "regulatory gaps," S. Rep. No. 94-698, at 1, through a comprehensive approach to chemical risk management that considered "the full extent of human or environmental exposure," H.R. Rep. No. 94-1341, at 6.

In amending TSCA in 2016, Congress sought to promote "effective implementation" of the 1976 law's objectives. See S. Rep. No. 114-67, 114th Cong., 1st Sess. (2015) at 2. At the time it strengthened TSCA, Congress affirmed that the intent of the original law—to give EPA "authority to look at the hazards [of chemicals] in total," S. Rep. No. 94-698, at 2—remained "intact." S. Rep. No. 114-67, at 7. Indeed, in a statement accompanying the law's passage, its Senate Democratic sponsors underscored that, with the expanded authorities conferred by Congress, TSCA should not be "construed as a 'gap filler' statutory authority of last resort" but "as the primary statute for the regulation of toxic substances."[9] Excluding all pathways of chemical exposure through air, water and soil from risk evaluations would be directly contrary to these Congressional expectations.

D. TSCA Section 9(b) Provides that EPA Must Decide Whether TSCA or Another Law is the Best Vehicle for Risk Management Only After Evaluating the Risks of a Chemical's Environmental Releases under TSCA

In the 1976 law, Congress recognized the need to coordinate use of TSCA with implementation of other environmental laws. However, it chose to do so not by excluding environmental releases from the purview of TSCA – the approach EPA is pursuing now. Instead, it established a framework for determining, on a case-by-case basis, whether the risks of particular chemicals are best addressed under these laws or under TSCA. Thus, section 9(b)(1) of TSCA provides that EPA may use TSCA regulatory authorities if it "determines, in [its] discretion, that it is in the public interest to protect against [a particular] risk by action taken under this Act" but should use other environmental laws if it determines

[9] Congressional Record – Senate 3517 (June 7, 2016).

that "a risk to health or the environment . . . could be reduced to a sufficient extent by actions taken under" these laws.

In 2016, Congress underscored the chemical-specific focus of this analysis by revising section 9(b)(2) so that, in deciding whether to regulate under TSCA or another law, EPA must "consider . . . all relevant aspects of the risk" in question and make a "comparison of the estimated costs and efficiencies" of addressing the risk under TSCA and other laws. Commenting on this language, the law's Senate Democratic sponsors explained that it allowed EPA to regulate under other laws in lieu of TSCA only where the "Administrator has already determined that a risk to health or the environment associated with a chemical substance or mixture could be eliminated or reduced to a sufficient extent by additional actions taken under other EPA authorities."[10]

This approach presupposes that EPA has already used the TSCA risk evaluation process to identify the risks of a chemical and the exposure pathways contributing to those risks and thus has an informed basis to determine whether they "could be eliminated or reduced to a sufficient extent" under another law. If EPA has not examined the specific pathways of environmental exposure and their contribution to total risk under TSCA, then it cannot conduct the analysis that section 9(b) requires because it will be unable to evaluate the relative strengths of using TSCA or another law to eliminate the risk. By presuming that other laws are *always* superior to TSCA in identifying and reducing the risks of chemicals in environmental media, EPA's blanket exclusion of environmental releases thus turns section 9(b) on its head.

E. Contrary to EPA, There is No Basis to Conclude that Other Environmental Laws are Equivalent in Scope and Protectiveness to TSCA

EPA's position that other environmental laws should displace TSCA risk evaluations for *all* chemicals arbitrarily assumes that these laws provide equivalent protection of public health and the environment and that there is no added benefit in addressing environmental pathways of exposure under TSCA. But in reality these other laws vary greatly in the degree of protection they afford against chemical risks and the extent of their application to unsafe chemicals. These limitations are precisely why Congress gave EPA comprehensive authority over chemical risks under TSCA in 1976 and strengthened that authority in 2016.

The 2016 TSCA amendments establish a risk-basic framework for EPA's decisions on chemical safety and set a high standard of protection of health and the environment. Under section 6(b)(4)(A), TSCA risk evaluations must: "determine whether a chemical substance presents an unreasonable risk of injury to health or the environment, *without consideration of costs or other non-risk factors*" (emphasis added). This determination must be for both the general population and "potentially exposed or susceptible subpopulations." Once an unreasonable risk is identified, TSCA section 6(c)(1) requires EPA to issue a rule under section 6(a) to address the risk. Section 6(a), in turn, directs that this rule must restrict the chemical "to the extent necessary so that the chemical substance no longer presents such risk" – again assuring protection of potentially exposed or susceptible subpopulations. As EPA has recognized, it

[10] Id.

cannot lower this level of protection based on consideration of costs and benefits.[11] Although the rule must be accompanied by an economic analysis, the restrictions it imposes must be sufficient to eliminate the unreasonable risk identified in the evaluation. Indeed, the 2016 TSCA revisions were explicitly designed to remove the cost-benefit framework required under the old law because it had impeded meaningful regulation of unsafe chemicals.[12]

TSCA's strict risk-based framework for chemical risk management is not mirrored in most environmental laws that govern releases to air, water and soil and disposal of waste. For example, the standard-setting process to establish discharge limits for chemical and other pollutants under the Clean Water Act (CWA) is technology-based and does not allow for consideration of risk.[13] The same is true of several provisions of the Clean Air Act (CAA) that regulate emissions from new and modified stationary sources of pollution and mobile sources.[14] In addition, the primary CAA mechanism for controlling industrial emissions of air toxics calls for EPA to set standards requiring Maximum Achievable Control Technology (MACT), an approach that does not take into account risks to health, although any "residual risks" can be addressed in a second stage of rulemaking.[15]

Even statutes that do allow for consideration of risks also direct EPA to weigh cost and other economic factors. The Safe Drinking Water Act (SDWA), for example, requires cost-benefit balancing in setting limits for drinking water contaminants, the very approach rejected in the 2016 TSCA amendments.[16] The Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA), which governs the remediation of contaminated sites, focuses on health protection but also directs EPA to take into account costs and technical achievability.[17] And importantly, most of these laws do not include TSCA's explicit protections for potentially exposed or susceptible subpopulations at higher risk than the general population. In short, the bulk of EPA-implemented environmental laws lack the high level of protectiveness and exclusive focus on eliminating unreasonable risks that Congress demanded in its recent TSCA revisions.

Equally important, in comparison to TSCA, the scope of regulation under other federal environmental laws is limited: these laws generally apply to only a subset of the substances that may present risks to health or the environment and only a subset of the facilities whose environmental releases contribute to these risks. For example, air toxics emission requirements in the CAA only address 189 Hazardous Air Pollutants (HAPs) designated by Congress in the 1990 CAA amendments[18] and only large industrial emitters that meet the CAA definition of "major source" are subject to emission limits.[19] Similarly, CERCLA cleanups encompass a statutory list of hazardous substances[20] and disposal requirements under

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[11] See proposed rule banning TCE use in vapor degreasing, 82 Fed. Reg. 7432, 7439-41 (Jan. 19, 2017).
[12] S. Rep. No. 114-67, at 4.
[13] 33 U.S.C. §1317.
[14] 42 U.S.C. §§7411,7475.
[15] 42 U.S.C. §7412.
[16] 42 U.S.C. §300g-1
[17] 42 U.S.C. §9621.
[18] 42 U.S.C. §7412(b).
[19] 42 U.S.C. §7412(a)(1).
[20] 42 U.S.C. §9601(14).

the Resource Recovery and Conservation Act (RCRA) only apply to those wastes that EPA has designated as "hazardous."[21] Industrial discharge limits under the CWA only apply to regulated "toxic" pollutants[22] and the CWA's water quality framework involves a complex mix of state and federal standards that vary across regions, may not address all pollutants that threaten human health and often do not result in uniform levels of protection. These basic gaps in coverage are painfully evident as EPA and states struggle to address widespread contamination and threats of harm to human health resulting from the extensive use and environmental release of Per- and polyfluoroalkyl substances (PFAS). Despite their significant risks, PFAS chemicals are not regulated as HAPs under the CAA, drinking water contaminants under the SDWA, hazardous substances under CERCLA or toxic pollutants under the CWA.

While EPA may have authority to expand the reach of its environmental laws to include previously unregulated toxics, it cannot do so without first evaluating the risks of these chemicals. With limited exceptions, however, EPA has no obligation under its environmental laws to assess the risks of unregulated chemicals or even to update its understanding of the hazard and exposure profile of those substances that are regulated. In practice, moreover, EPA's other regulatory programs have limited resources and many competing priorities, including those required by specific statutory provisions and/or court orders. Thus, there is little likelihood that previously unaddressed chemical risks will be evaluated by these programs. Indeed, many existing environmental standards are decades old and no longer reflect the best available science but EPA's environmental media programs lack the bandwidth and inclination to update them based on current understanding of risks to human health and the environment. For all these reasons, by precluding the use of TSCA to determine the health and environmental impacts of chemical releases to air, water and soil, EPA is effectively closing the door to any meaningful evaluation of these impacts – and, thus, to the use of TSCA or other laws to restrict those releases that are found to be unsafe.

In sum, exclusion of all environmental releases from TSCA risk evaluations is contrary to the wording, intent and purposes of the law and will inevitably mean that serious threats to health and the environmental are neither identified nor addressed.

III. There is No Legal or Technical Justification for Excluding General Population Exposure from EPA's Risk Evaluations

Several of the problem formulations indicate that EPA will not evaluate the risks of general population exposure. As stated in the PERC problem formulation:

> EPA does not plan to consider and analyze general population exposures in the risk evaluation for PERC. EPA has determined that the existing regulatory programs and associated analytical processes have addressed or are in the process of addressing potential risks of TCE that may be present in various media pathways (e.g., air, water, land) for the general population. For these cases, EPA believes that the TSCA risk evaluation should focus not on those exposure pathways,

[21] 42 U.S.C. §6921.
[22] 42 U.S.C. §1317(a).

but rather on exposure pathways associated with TSCA uses that are not subject to those regulatory processes.[23]

This approach is unjustified for the reasons discussed above. If the presence of a chemical in environmental media – and therefore exposure to the chemical by the general population – is attributable to its "conditions of use", there is no basis for excluding this background level of exposure from EPA's risk evaluation. The claim that this exclusion is justified because "existing regulatory" programs apply to environmental releases is unsupported by the law: in accordance with section 9(b), EPA must first determine the risk resulting from environmental releases through a TSCA risk evaluation and then determine whether the risk is best addressed under TSCA or other EPA-administered environmental laws.

The goal of risk evaluations under section 6(b)(4)(A) is to determine the risks presented by a chemical as a whole, not the risks of individual uses and pathways in isolation. Moreover, section 6(b)(4)(F) directs EPA to take into account "the likely duration, intensity, frequency and number of exposures under the conditions of use of the chemical substance" and to "integrate and assess available information on hazards and exposures for the conditions of use." This integrating analysis cannot be performed if some pathways of exposure are excluded simply because they involve environmental media and could be subject to other laws. As the House Report for original TSCA emphasized, "[i]ntelligent standards for regulating exposures to a chemical in the workplace, the home or elsewhere in the environment cannot be set unless the full extent of human or environmental exposure is considered."[24]

The background levels of a chemical in the environment may present an unreasonable risk to the general population in their own right or they may add to other sources of exposure to present an overall risk to specific populations that is unreasonable. In either event, EPA cannot discharge its obligations under the law unless it determines and takes into account the background levels of a chemical to which the general population is exposed.

IV. EPA's Continues to Fail to Explain What Methodology It Will Use to Account for Multiple Exposure Pathways that Increase Overall Risk

The law's clear requirements for evaluating and protecting against risks to "potentially exposed or susceptible subpopulations" further underscore EPA's obligation to consider all contributors to exposure and risk, including a chemical's presence in environmental media. In order to determine whether a subpopulation may be at greater risk because it has greater exposure than the general population, the Agency must first quantify general population exposure and then determine how this exposure is increased because of exposures in the workplace, through products, as a result of environmental releases or because of other pathways that affect a particular subpopulation. To protect these subpopulations, EPA's focus must be on whether the total risk they face, considering all sources of exposure, is unreasonable. If one or more contributors to exposure are ignored, groups who are at

[23] Problem Formulation of the Risk Evaluation for Perchloroethylene (May 2018) at 73.
[24] House Rept. No. 94-1341, supra, at 6.

greater risk than the general population because of multiple exposure pathways will be inadequately protected.

Recognizing the need to account for the impact of multiple sources of exposure, TSCA section 6(b)(4)(F)(ii) requires risk evaluations to describe whether aggregate or sentinel exposures to a chemical were considered and the basis for that consideration. To properly apply either or both of these approaches in a risk evaluation, EPA must determine in advance what methodology it will employ and then incorporate it in the risk evaluation design in sufficient detail to describe the key data sources it will use to assess exposure and how they will be used.

EPA has not done this. Disappointingly, neither the scoping documents nor the problem formulations shed any light on how EPA risk evaluations will account for multiple pathways of exposure by the general population or subpopulations. Instead, it appears that EPA will examine each source of exposure in isolation and will not consider either the combined effect of multiple exposures or the contribution of environmental releases to overall exposure and risk. This is a violation of TSCA.

V. Ongoing Use and Disposal of Chemical Products that are No Longer Being Manufactured Fall Within the TSCA Definition of "Conditions of Use" and Cannot Be Excluded from Risk Evaluations

Among the 10 chemicals are substances, such as asbestos and HBCD, that contribute to ongoing exposure and risk as a result of historical manufacturing and processing activities that have been discontinued. In many cases, the current and foreseeable risks associated with these activities are significant. Nonetheless, the problem formulations, like the scoping documents, take the position that they are outside the scope of risk evaluations. As stated in EPA'S asbestos problem formulation:

> In the case of asbestos, legacy uses, associated disposals, and legacy disposals will be excluded from the problem formulation and risk evaluation, as they were in the Scope document. These include asbestos containing materials that remain in older buildings or are part of older products but for which manufacture, processing and distribution in commerce are not currently intended, known or reasonably foreseen. EPA is excluding these activities because EPA generally interprets the mandates under section TSCA § 6(a)-(b) to conduct risk evaluations and any corresponding risk management to focus on uses for which manufacture, processing or distribution is intended, known to be occurring, or reasonably foreseen, rather than reaching back to evaluate the risks associated with legacy uses, associated disposal, and legacy disposal, and interprets the definition of conditions of use in that context. [25]

EPA is incorrectly interpreting the provisions of LCSA. The definition of "conditions of use" in section 3(4) includes the "circumstances . . . under which a chemical substance is . . . known or reasonably foreseen to be . . . used or disposed of." Where a chemical is performing an ongoing *in situ* function as a result of

[25] Problem Formulation of the Risk Evaluation for Asbestos (May 2018) at 8.

previous manufacturing and processing activity, that function comprises a current "use" of the chemical that is "known" to be occurring.[26]

For example, although asbestos may no longer be sold as insulation, the asbestos insulation installed in millions of US buildings continues to perform insulating functions and thus is a current ongoing "use" of asbestos. Installed asbestos-containing building materials (ACBMs) represent one of the largest sources of asbestos accessible to the general public in the US, and the largest asbestos-exposed population consists of people who occupy buildings and homes with ACBMs. Maintenance and construction activities involving ACBMs are also frequent and widespread and account for the largest present-day increase in mesothelioma illness and death in the US.[27]

Similarly, the Healthy Building Network estimates there are 66 million- 132 million pounds (30,000-60,000 metric tons) of HBCD in insulation in existing buildings.[28] These ongoing insulation uses are and will continue to be critical sources of ongoing exposures. HBCD is also present in cars and furniture as a flame retardant and its use in these long-lived consumer articles will contribute to ongoing exposures for years to come.[29]

Equally important, the disposal of building materials or consumer products containing asbestos or HBCD is an ongoing occurrence as buildings are torn down or remodeled and cars and furniture are replaced. Thus, the resulting releases into the environment and communities comprise a "circumstance . . . under which [these chemicals] are . . . known or reasonably foreseen to be . . . disposed of." As "conditions of use" within the TSCA definition, these activities and the risks they present are likewise required to be addressed in risk evaluations under section 6(b). For both chemicals, the immediate and long-term exposures associated with disposal of *in situ* building materials and products are likely to be widespread and significant well into the future.[30]

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[26] SCHF and its co-petitioners are challenging EPA's position that ongoing use and disposal of discontinued products are not TSCA "conditions of use" in *Safer Chemicals Healthy Families v. EPA*, 17-72260 (9th Cir.) In addition to being used and disposed of, legacy products that perform functions in the built environment can be considered "distributed in commerce" as this term is defined in TSCA section 3(5). The definition includes "to hold, or the holding of, the substance, mixture or article after its introduction in commerce" – language that plainly applies to *in situ* products. Likewise, the definition includes the "introduction or delivery for introduction into commerce" of the substance, mixture or article. This description would apply to legacy products that are repurposed or sold for recycling.

[27] US CDC study, "Malignant Mesothelioma Mortality – United States 1999 to 2005."

[28] Safer Chemicals, Healthy Families et al. Comments to the U.S. Environmental Protection Agency (EPA) on the Scope of its Risk Evaluation for the TSCA Work Plan Chemicals: CYCLIC ALIPHATIC BROMIDE CLUSTER or HEXABROMOCYCLODODECANE (HBCD). March 15, 2017. https://healthybuilding.net/uploads/files/saferchemicals-hbcd.pdf

[29] It is unclear whether EPA intends to exclude installed HBCD-containing building and construction materials from its risk evaluation. The problem formulation states that the evaluation will address "commercial/consumer use" of "building/construction materials" but this could be interpreted to apply to materials that are available for use in ongoing construction projects and not those already installed. See Problem Formulation for Cyclic Aliphatic Bromides Cluster (HBCD) (May 2018) at 29.

[30] EPA also excludes disposal from the asbestos and HBCD risk evaluations based on its overall determination that the release of chemicals to environmental media should not be addressed under TSCA. Oddly, disposal of HBCD construction and demolition waste is listed as a condition of use EPA plans to address in one part of its problem

To exclude from risk evaluations ongoing and future exposures from *in situ* uses of discontinued products would create a sizable gap in the life-cycle assessments of risk that Congress directed EPA to conduct under the new law. This would deprive the public, scientists and regulators of a comprehensive picture of one of the largest sources of continuing and future risk. Since *in situ* sources of exposure form a critical component of the background levels of asbestos and other chemicals to which the general population is exposed, EPA's assessment of risks to particular subpopulations from more specific exposure pathways would also be incomplete and understated.

In addition, decision-makers would be unable to reduce ongoing exposures and impose safeguards against unsafe use and disposal and "legacy" products because they would lack a meaningful risk evaluation to inform these actions. Just as TSCA provides authority to evaluate the risks associated with ongoing exposures from discontinued activities, so it gives EPA the authority under section 6(a) to reduce these risks, yet the Agency would be stymied by the absence of a risk evaluation that provides a basis for such regulation.[31]

In short, EPA must characterize and assess ongoing exposures from the use and disposal of discontinued products and determine the risks they present as part of its risk evaluations on the initial 10 chemicals. Its continuing failure to do so is a clear violation of TSCA.

VI. Uses Discontinued under the Threat of Regulatory Action Fall Within the TSCA Definition of "Conditions or Use" and Must be Addressed in TSCA Risk Evaluations

A number of the problem formulations indicate that certain chemical uses have been discontinued and therefore will not be addressed in the risk evaluation for that chemical.

The problem formulation for HBCD illustrates this approach. Based on representations by industry, EPA asserts that HBCD use in the production of flame retardants, EPS resins, high impact polystyrene, XPS master batch, motor vehicle upholstery, consumer textiles, and military, institutional and aviation textile applications has ceased. According to EPA, these uses are no longer "intended, known or reasonably foreseen" and therefore do not comprise TSCA "conditions of use" that will be addressed in the HBCD risk evaluation.[32] EPA also indicates that because HBCD is no longer being manufactured in the US, domestic production will likewise not be addressed.

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formulation (page 29) but then identified as an exposure pathway that will not be considered later in the same document (page 52).

[31] For some chemicals like lead and asbestos, other laws administered by EPA address handling and disposal of *in situ* materials and the Agency may be able to refer the findings of its risk evaluations to the programs implementing these laws under TSCA section 9(b) in lieu of further regulation under section 6. However, there are no existing laws that address ongoing exposure from use and disposal of discontinued products containing HBCD, perfluorinated chemicals and other substances and therefore the availability of the protections afforded under section 6 of TSCA may be critical to addressing their risks. Obviously, if these risks are not identified and evaluated under TSCA section 6(b), there will be no basis for reduction them through regulation under section 6(a).

[32] Problem Formulation of the Risk Evaluation for Cyclic Aliphatic Bromides Cluster (HBCD), at 24-25.

EPA has not disclosed the industry communications it is relying on but it appears they are informal and non-binding and have not been verified by the Agency. Nor has EPA indicated that it has contacted all HBCD producers and users to confirm that the uses in question have been fully eliminated. Thus, there is no assurance that these HBCD uses no longer exist and, if so, will not be revived in the future. Indeed, the most likely explanation for the phase-out of previously well-established HBCD uses is the regulatory and public scrutiny HBCD has received, a consideration that could wane in importance in the future, particularly if the risks presented by these uses are not evaluated or restricted by EPA.

EPA has also narrowed the scope of the asbestos risk evaluation by excluding now discontinued but historically significant asbestos-containing products and failing to address mining of asbestos in the US. Instead, EPA has proposed a significant new use rule (SNUR) so that it is notified of the reintroduction of discontinued products before it occurs.[33] However, while EPA has the ability to ban or restrict a new use after receiving notification under a SNUR, the SNUR does not itself comprise a finding of unreasonable risk nor does it provide any assurance that the use would be regulated once the Agency receives a significant new use notice (SNUN). With the exclusion of discontinued asbestos uses, the EPA risk evaluation will be limited to the small number of asbestos products that remain in commerce, providing a grossly incomplete picture of the threat to health from past and potential future uses of asbestos.

We disagree with EPA that discontinuance of a previously widespread use necessarily places it beyond the reach of section 6 risk evaluation and management authorities. EPA provides no justification for its assertion that the TSCA definition of "conditions of use" does not apply to such uses. As defined in section 3(4), this term includes not simply intended or known uses but the "circumstances under which a chemical substance is . . . reasonably foreseen to be manufactured, processed, distributed in commerce, used or disposed of." It is clearly "reasonably foreseen" that long-standing and significant uses of a chemical that have been phased out may re-enter commerce in the absence of any legal restriction. Moreover, section 6(a) provides that EPA must regulate a chemical where "manufacture, processing, distribution in commerce, use or disposal" presents an unreasonable risk but does not stipulate that these activities must be currently occurring to warrant restriction. Indeed, the purpose of section 6(a) rules – to impose the measures "necessary so that the chemical substance no longer presents [an unreasonable] risk" – is equally applicable to ongoing commercial activities and to historical uses that could resume and require restrictions so they do not cause harm to health and the environment.

Although the 2016 TSCA amendments removed the phrase "will present" from section 6(a), the statement of Democratic sponsors at the time of enactment makes clear that EPA retained its authority to address anticipated future risks:

> "Existing TSCA as in effect before the date of enactment of Frank R Lautenberg Chemical Safety for the 21st Century Act includes the authority, contained in several sections (see, for example, section 6(a)), for EPA to take regulatory actions related to chemical substances or mixtures if it determines that the chemical substance or mixture 'presents or will present' an unreasonable risk to health or the environment. The Frank R. Lautenberg Chemical Safety for the 21st Century

[33] 83 Fed. Reg. 26922 (June 11, 2018).

> Act includes language that removes all instances of 'will present' from existing TSCA and the amendments thereto. *This does not reflect an intent on the part of Congressional negotiators to remove EPA's authority to consider future or reasonably anticipated risks in evaluating whether a chemical substance or mixture presents an unreasonable risk to health or the environment. In fact, a new definition added to TSCA explicitly provides such authority and a mandate for EPA to consider conditions of use that are not currently known or intended but can be anticipated to occur . . .* "[34]

The goals of TSCA would be defeated if manufacturers of unsafe chemicals could avoid scrutiny simply by ceasing production for specific uses before EPA completes a risk evaluation of those uses and then later re-entering the marketplace free from any restriction or determination of risk. This scenario is particularly troubling where the product phase-out is in response to agency risk concerns and intended to avoid the consequences of an adverse risk finding and subsequent regulatory action. In these cases, the best interpretation of TSCA is to treat the possible reintroduction of a discontinued use as "reasonably anticipated," to address that use in the risk evaluation and to then ban or restrict it permanently under section 6(a) if it is determined to present an unreasonable risk.

We do not believe a SNUR is an adequate substitute for evaluation and regulation of a discontinued chemical use under section 6. SNURs are fundamentally notification requirements and do not themselves require an assessment or determination of risk. The activities they define as "significant new uses" are not prohibited: companies seeking to conduct these activities must notify EPA at least 90 days before initiating them. While the Agency must review the new use and ban or restrict it under sections 5(e) or 5(f) upon determining that the use does or may present an unreasonable risk, the Agency may or may not choose to take these actions. Thus, the door will not be closed to reintroduction of the use. Moreover, EPA's review of a SNUN and decision to regulate the new use lack the elements of openness and accountability that apply during section 6 risk evaluations and rulemakings. Thus, these decisions will receive limited public and judicial review.

A comprehensive risk evaluation under section 6, by contrast, enables the Agency to make a definitive risk determination for plausible future risk scenarios in a transparent process that provides clarity to industry and the public and closes the door to the resumption of unsafe uses. If there is a role for a SNUR, it is to perform the limited stop-gap function of assuring that EPA is notified of significant changes in use while its risk evaluation and follow-up rulemaking are underway so that these uses are not re-established in the marketplace before EPA has addressed their risks under section 6 and restricted them if warranted.

VII. EPA Should Not Make Determinations of Unreasonable Risk for Endpoints that Lack Adequate Information and Should Use its Section 4 Authorities to Require Industry to Fill These Data gaps

[34] Cong. Record – Senate 3515 (June 7, 2016) (emphasis added).

Our groups have repeatedly called for EPA to identify data gaps that limit its ability to reach definitive conclusions about the health and environmental effects of the 10 chemicals.[35] We have urged EPA to take steps to fill these data gaps early in the risk evaluation process using its expanded TSCA information development authorities so that sufficient information is available for an informed evaluation. EPA itself has emphasized the need for comprehensive data on hazard and exposure before it initiates evaluations although it has backed away from a systematic information collection process at the pre-prioritization stage for risk evaluation candidates.[36] Basing risk evaluations on adequate data is not only necessary to meet EPA's obligation under section 26(k) to consider all "reasonably available information" but furthers section 2(b)(2), which declares that "[i]t is the policy of the United States" that "adequate data should be developed with respect to the effect of chemical substances and mixtures on health and the environment."

It is therefore disappointing that the problem formulations, like the earlier scoping documents, make minimal efforts to identify significant data gaps for the 10 chemicals, to set in motion development of additional information, and to address how these data gaps will impact the conclusions reached in the risk evaluations. Indeed, EPA seems ready to find that substances do not present an unreasonable risk of injury even where available data are lacking entirely or are insufficient under Agency guidelines to determine that a substance lacks adverse effects.[37]

Pigment violet 29 is a case in point. The problem formulation for this substance indicates that, based on the absence of significant evidence of hazard, EPA "expects to be able to reach conclusions about particular conditions of use, hazards, or exposure pathways without further analysis."[38] Yet nowhere does EPA address whether it has sufficient information to reach such conclusions for major health end-points. EPA's Design for the Environment (now known as Safer Choice) Program and risk evaluation

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[35] See, e.g., Comments of Safer Chemicals Healthy Families on Proposed Procedures for Chemical Risk Evaluations under the Amended Toxic Substances Control Act Submitted via Regulations.gov (March 20, 2017), Docket ID EPA-HQ-OPPT-2016-0654

[36] In the discussion paper EPA prepared for its December 11, 2017 public meeting on prioritization, EPA stated that:

> Prior to designating a chemical as a high-priority for risk evaluation, it is important for EPA to ensure the reasonably available information is sufficient to conduct a scientifically robust risk evaluation. In many cases, EPA believes it would be difficult to require the development of necessary chemical substance information, evaluate that information, and incorporate that information into analyses and decisions within the statutory timeframes associated with the prioritization and risk evaluation processes. Therefore, it will be useful for EPA to identify information needs and determine whether any of these needs should be addressed before initiating the prioritization process.

DISCUSSION DOCUMENT: Possible Approaches and Tools for identifying Possible Candidate Chemicals for Prioritization at 7. Despite this recognition, EPA's final prioritization framework rule deleted a pre-prioritization process that would have expressly provided a process for identifying and filling data gaps before risk evaluations are initiated. *Procedures for Prioritization of Chemicals for Risk Evaluation under the Toxic Substances Control Act.* 82 Fed. Reg. 33753 (July 20, 2017).

[37] The EPA responses to comments on the scoping documents indicate that: "when OPPT does find existing data are not adequate, OPPT will use all available authorities to fill data gaps necessary to conduct fit-for-purpose assessments." This is not, however, the approach reflected in the problem formulations.

[38] Problem Formulation of the Risk Evaluation for Pigment Violet 29 (May 2018) at 7.

guidelines and REACH requirements in the EU identify the studies deemed necessary for an informed risk evaluation. The database for pigment violet 29 is deficient when measured against these authoritative sources. Illustrating these deficiencies, the table below compares the test data available on pigment violet 29 with the requirements for a DfE/Safer Choice human health hazard trait assessment. [39]

DfE Hazard Trait	**Empirical Data Available for Pigment Violet 29?**[40]
Acute mammalian toxicity	Yes. In vivo oral, dermal and inhalation acute toxicity studies are available, though the inhalation studies are deemed to be unsuitable by ECHA.[41]
Respiratory sensitization	No
Skin sensitization	Yes, in vivo study
Eye irritation/ corrosivity	Yes, in vivo study
Skin irritation/ corrosivity	Yes, in vivo study
Carcinogenicity	No
Mutagenicity/ genotoxicity	Yes. In vitro gene mutation and mammalian cells genetic toxicity studies available.
Reproductive and developmental toxicity	Yes, screening study
Developmental neurotoxicity	No
Neurotoxicity	No
Repeated dose toxicity	No
Endocrine activity	No

Thus, EPA could not reach scientifically defensible conclusions that pigment violet 29 lacks the potential to cause carcinogenicity, reproductive and developmental toxicity, developmental neurotoxicity, neurotoxicity, repeated dose toxicity or endocrine effects.

[39] SCHF is grateful to scientists at the University of California San Francisco for preparing this table, which is included in comments on the problem formulations filed on behalf of a group of academics, scientists, and clinicians.

[40] Information from: US EPA (May 2018) Problem Formulation of the Risk Evaluation for Pigment Violet 29. European Chemicals Agency (ECHA). (2017). Perylene-3, 4; 9, 10-tetracarboxydiimide. Helsinki, Finland. Available: https://echa.europa.eu/registration-dossier/-/registered-dossier/10330

[41] ECHA states: "Unsuitable test system, as the inhalation hazard test is insufficient for non-volatile substances." Available: https://echa.europa.eu/registration-dossier/-/registered-dossier/10330/7/3/3/?documentUUID=34aa4522-b714-47b0-9bee-af8052fff73d

Pigment violet 29 is not the only one of the 10 chemicals with significant data gaps. 1,4-dioxane, MC, PERC and TCE also lack data for important end-points:

1,4 Dioxane. For this chemical, there is little or no information on the potential for developmental toxicity or developmental neurotoxicity. This is especially problematic given that the chemical is a well-known neurotoxic agent. This critical data gap was identified by ATSDR in its 2012 Tox Profile.[42]

MC. MC is a known human neurotoxicant, associated with depression of the central nervous system, and severe dose-dependent neurotoxic effects including headaches, slowed reaction time, decreased alertness, impaired movements, loss of consciousness, coma, seizures, and death. (It has been shown in animal studies to cross the placenta, and in humans it has been detected in breast milk.[43]) Yet, the chemical has not been adequately tested for developmental neurotoxicity. This is especially alarming given the widespread use and population exposure to this deadly neurotoxic chemical. Chemicals that are neurotoxic should be presumed to be developmentally neurotoxic. That is, compared with adult exposures, they are much more damaging and at much lower levels when exposures take place during early fetal development.[44] The failure to test and appropriately regulate these chemicals has led to debilitating neurodevelopmental disorders such as autism, learning deficits, and behavioral problems – all with disastrous impacts on affected individuals, families, and society.

PERC. This chemical is considered by EPA to be both neurotoxic and a developmental toxicant, yet it has never been tested for developmental neurotoxicity. This is a major data gap, given that developmental neurotoxic effects such as learning impairments and behavioral problems are often overlooked in routine tests such as the ones EPA considered, which focus on crude frank toxicity such as reduced body or organ weights, stillbirths and deaths (see Perc problem formulation, p. 52). Lead, mercury, and other developmental neurotoxic chemicals have all been shown to have virtually no safe level when exposures occur prenatally during critical windows of neurodevelopment.[45] For this reason, the EPA pesticide office began requiring pesticide registrants to submit developmental neurotoxicity testing – which includes subtle but important endpoints like motor activity, learning and memory, and auditory startle response – for the organophosphates and other pesticides known to be neurotoxic.[46] In an EPA fact sheet issued last month, EPA emphasizes why specific developmental neurotoxicity tests are important:[47]

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[42] Agency for Toxic Substances and Disease Registry (ATSDR). 2012. Toxicological profile for 1,4 Dioxane. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. P. 143. https://www.atsdr.cdc.gov/toxprofiles/tp.asp?id=955&tid=199

[43] ATSDR Medical Management Guidelines for Methylene Chloride. Updated 2014. https://www.atsdr.cdc.gov/MMG/MMG.asp?id=230&tid=42

[44] Grandjean P, Landrigan PJ. Developmental neurotoxicity of industrial chemicals. Lancet. 2006 Dec 16;368(9553):2167-78. Review.

[45] Grandjean P, Landrigan PJ. Developmental neurotoxicity of industrial chemicals. Lancet. 2006 Dec 16;368(9553):2167-78. Review.

[46] EPA OPPTS 870.6300 Developmental neurotoxicity study. 1996. EPA 712-C-96-239.

[47] EPA Science Brief. Evaluating Developmental Neurotoxicity. July 2018. https://www.epa.gov/sites/production/files/2018-07/documents/dnt_factsheet_07_23_18_final.pdf

- The developing nervous system can be particularly sensitive to exposure to environmental chemicals.
- Less than 1% of chemicals in the environment have been fully evaluated for their potential to be developmental neurotoxicants, or their impact on the developing nervous system.
- Due to a lack of data, it is not possible to understand the extent or potential contribution of environmental chemicals in neurodevelopmental disease, nor predict the potential developmental neurotoxicity risk for individual chemicals.

The failure to address the risks of developmental neurotoxicity posed by PERC represents a serious data gap in EPA's assessment, particular for the low-dose risks.

TCE. Trichloroethylene was evaluated well over a decade ago, in 2004, by the EU, which at the time identified the need for developmental neurotoxicity testing to be conducted for TCE:

> The developmental toxicity of inhaled trichloroethylene at non-maternally toxic levels (up to 1,800 ppm) has been investigated in rats, mice and rabbits in conventional studies. No evidence of developmental toxicity was reported. In contrast, the results of a series of non-standard oral studies in rats raised some concerns about the potential for trichloroethylene to induce developmental neurotoxicity at dose levels in the range of 30-110 mg/kg/day. However, these studies were of limited scope and were considered not to provide sufficient basis on which to draw clear conclusions about the hazardous properties of trichloroethylene. To be able to draw clear conclusions regarding developmental neurotoxicity, further testing according to the draft OECD TG 426 Developmental Neurotoxicity guideline would be required."[48]

The 2011 IRIS assessment comes to similar conclusions, also identifying the potential for developmental neurotoxicity and noting this data gap:

> In summary, an overall review of the weight of evidence in humans and experimental animals is suggestive of the potential for developmental toxicity with TCE exposure. A number of developmental outcomes have been observed in the animal toxicity and the epidemiological data, as discussed below. These include adverse fetal/birth outcomes including death (spontaneous abortion, perinatal death, pre- or post-implantation loss, resorptions), decreased growth (low birth weight, SGA [small for gestational age], IUGR [intrauterine growth restriction], decreased postnatal growth), and congenital malformations, in particular cardiac defects. Postnatal developmental outcomes include developmental neurotoxicity, developmental immunotoxicity, and childhood cancer.[49]

The TCE problem formulation identifies the risk of neurotoxicity and developmental toxicity separately, noting evidence from both human studies and animal studies, including psychomotor effects from TCE exposures.[50] Yet, there is no study that specifically targets the sensitive and critical endpoint of

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[48] European Union 2004, Risk Assessment Report for Trichloroethylene, p. 241. https://echa.europa.eu/documents/10162/83f0c99f-f687-4cdf-a64b-514f1e26fdc0

[49] EPA 2011, Toxicological Review of Trichloroethylene for IRIS, available at: http://www.epa.gov/iris/supdocs/0199index.html, p. 4-556

[50] EPA 2018 TCE Problem Formulation p. 45. See also the EPA IRIS 2011 Toxicological Review of Trichloroethylene.

developmental neurotoxicity. The failure to address the risks of developmental neurotoxicity posed by TCE represents a serious data gap in EPA's assessment, particular for the low-dose risks.

In the face of material data gaps, an unqualified conclusion that a chemical does not "present an unreasonable risk of injury" to health could not be defended under TSCA and would misinform the public about the chemical's safety.[51] Thus, EPA's risk evaluations should be explicit about the health and environmental end-points that lack adequate data and should exclude these end-points from its determinations of unreasonable risk. It should also use its TSCA authorities to require manufacturers to conduct testing to develop adequate data for a defensible risk evaluation so that future assessments can be informed by a comprehensive dataset. EPA's lack of interest in using section 4 of the law to generate data necessary for risk evaluation is deeply troubling in light of the clear intent of the 2016 TSCA amendment to provide the Agency with the tools to require more testing by industry to support priority setting and risk evaluations under section 6.

VIII. Where EPA Believes that Particular Conditions of Use Present *De Minimis* Risks, It Cannot Drop These Uses with no Additional Analysis, But Rather Must Explain and Document Why Their Risks Are Insignificant

The problem formulations also indicate that EPA "expects to be able to reach conclusions about particular conditions of use, hazards or exposure pathways without further analysis" and will not further address them in its risk evaluations.[52] For example, EPA indicates that it will devote no further attention to multiple uses of carbon tetrachloride (CTC) that it asserts pose only *de minimis* risks:

> Because industrial, commercial, and consumer use of such products (solvents for cleaning/degreasing, adhesives/sealants, and paints/coatings) would present only de minimis exposure or otherwise insignificant risk, EPA has determined that these conditions of use do not warrant evaluation, and EPA does not expect to consider or evaluate these conditions of use or associated hazards or exposures in the risk evaluation for carbon tetrachloride.[53]

Nowhere has EPA provided general criteria for determining levels of exposure or risk that are "insignificant" for purposes of TSCA risk evaluations. Nor has the Agency explained why it considers carbon tetrachloride-containing solvents with potential consumer, industrial and commercial exposure to be so inconsequential that they can be determined not to present "unreasonable risks" without any product-specific analysis of use and release scenarios.[54] Since carbon tetrachloride is a carcinogen, even

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[51] EPA has recognized that "OPPT does not believe that absence of data equals no risk." EPA's Responses to Public Comments Received on the Scope Documents for the First Ten Chemicals for Risk Evaluation under TSCA (May 2018) at 13. However, the problem formulations suggest that the Agency is not applying this principle in its evaluations of individual chemicals.

[52] This statement appears in the Introduction to all of the Problem Formulations. See, e.g., Problem Formulation of the Risk Evaluation for Carbon Tetrachloride at 13.

[53] Id., at 21.

[54] EPA's initial use summary found products with up to 2.5% CTC and SCHF's submission to EPA of publically available product information included products with 1% CTC. See Safer Chemicals, Healthy Families,

low concentrations cannot be assumed to be safe without some understanding of the conditions and levels of exposure. Moreover, even if the risk from a specific product is small in itself, multiple products and exposure pathways may result in aggregate levels of exposure that present significant risks to one or more worker or consumer subpopulations. As noted above, TSCA requires EPA to examine chemical risks holistically, taking into account all uses and pathways of exposure, and cannot summarily eliminate an entire class of products from consideration. EPA may have some latitude to devote greater effort to some exposure and risk scenarios than others, but this does not excuse ignoring particular conditions of use based on the unsubstantiated claim that they present *de minimis* risks.

It is also troubling that, despite numerous critical comments, EPA continues to ignore the presence of 1,4-dioxane as an impurity in products on the ground that "contamination of industrial, commercial and consumer products are not intended conditions of use for 1,4-dioxane and will not be evaluated."[55] EPA's position is legally unsupportable. Production of a chemical as a byproduct or impurity is plainly a "circumstance . . . under which a chemical substance . . . is known . . . to be manufactured" and thus falls squarely within the definition of "conditions of use" in section 3(4) of TSCA. There is no basis in this provision or other parts of the law for differentiating between manufacture as a byproduct/impurity and purposeful production and including the latter in a risk evaluation but excluding the former. In the case of 1,4-dioxane, EPA has made no effort to argue that byproduct/impurity production poses *de minimis* risks and such a position could not be defended given the evidence that 1,4-dioxane's detection in drinking water and groundwater is linked in part to its presence as a contaminant in products and waste streams released into the environment. Plainly, EPA must add 1,4-dioxane production as a byproduct and impurity to the scope of its risk evaluation.

IX. EPA Cannot Drop Significant Hazards from Risk Evaluations

The asbestos problem formulation provides another example of an EPA decision "not to further analyze" a potential source of risk. EPA has chosen to limit its asbestos evaluation to lung cancer and mesothelioma.[56] Yet the asbestos scoping document is clear that several other cancers have been linked to asbestos: [57]

> Mortality studies of asbestos workers have revealed increases in cancer mortality at one or more sites other than the lung, the pleura or the peritoneum. Cancer of the larynx and ovary and gastrointestinal cancers, such as colorectal, pharynx and stomach, have been observed in populations exposed to various types of asbestos (IARC, 2012; NRC, 2006). Some studies have also noted excess deaths from, or reported cases of, cancers at other sites, such as the kidney and esophagus; however, the evidence is not consistent.

Non-malignant diseases are also caused by asbestos, including asbestosis and asbestos-related pleural thickening.

Environmental Health Strategy Center, Healthy Building Network, Comments to the U.S. Environmental Protection Agency (EPA) on the Scope of its Risk Evaluation for the TSCA Work Plan Chemical: CARBON TETRACHLORIDE (CTC) CAS Reg. No. 56-23-5 (March 15, 2017). This information is not reflected in the problem formulation for CTC.

[55] Problem Formulation of the Risk Evaluation for 1,4-Dioxane (May 2018) at 18.

[56] Problem Formulation of the Risk Evaluation for Asbestos at 34.

[57] Scope of the Risk Evaluation for Asbestos (May 2017) at 34-35.

The comprehensive approach to risk evaluations in TSCA requires EPA to address all known hazards of a chemical, particularly one whose dangers to human health are so serious and well documented. The law provides no basis for failing to evaluate documented adverse health effects, let alone effects of this severity and magnitude.

X. EPA Should Not Revisit Definitive Findings in IRIS Assessments Unless There Are New Data That Inform EPA's Evaluation of the Weight of the Evidence

Six of the 10 chemicals -- asbestos, TCE, MC, CTC, PERC and 1,4-dioxane -- have been assessed under the EPA Integrated Risk Information System (IRIS). The IRIS process is the Agency's authoritative mechanism for reviewing available studies, characterizing the health effects of chemicals and identifying concentrations below which these chemicals are not likely to cause adverse effects. IRIS assessments typically reflect years of work by EPA scientists, multiple rounds of public comment, inter and intra-agency consultation, and extensive peer review, often by the Agency's independent Science Advisory Board (SAB) or the National Academy of Sciences (NAS). The IRIS program recently received a favorable review from the NAS.[58]

Where EPA is conducting a TSCA risk evaluation of a chemical that has already been assessed under IRIS, the conclusions of the IRIS assessment should be presumed to be applicable to the TSCA evaluation as a definitive statement by the Agency of the best available science. Reopening IRIS findings would harm the public by prolonging uncertainty on issues that have been addressed and resolved through an authoritative, transparent and inclusive EPA process. Like other Agency actions, IRIS assessments often give rise to differences of opinion and some stakeholders may be disappointed by the outcome. But this does not mean that EPA should reinvent the wheel and provide another bite at the apple on scientific determinations that have been made after thorough deliberation. To revisit IRIS findings would also be inefficient and resource-intensive at a time when the Agency is struggling with workforce and budget constraints and is straining to manage its TSCA workload.

The only rationale for revisiting IRIS findings is where significant new data have become available since the final IRIS assessment that could inform the weight of the evidence on particular end-points. If that is the case, then the IRIS program should be tasked with updating its previous assessment, using a systematic review protocol that is consistent with the state of the science such as the National Toxicology Program (NTP) method.[59] In its response to comments on the scoping documents, EPA seems to adopt this limited approach to reopening IRIS conclusions, stating that:

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[58] National Academies of Sciences, Engineering, and Medicine. 2018. Progress Toward Transforming the Integrated Risk Information System (IRIS) Program: A 2018 Evaluation. Washington, DC: The National Academies Press. https://doi.org/10.17226/25086.

[59] National Toxicology Program. Handbook for Conducting a Literature-Based Health Assessment Using OHAT Approach for Systematic Review and Evidence Integration. In: U.S. Department of Health and Human Services, editor.: Office of Health Assessment and Translation, Division of National Toxicology Program, National Institute of Environmental Health Sciences; 2015

> OPPT has used IRIS documents as a starting point for identifying key and supporting toxicity studies and initial hazard identification. However, EPA also expects to consider other available hazard and exposure data to ensure that all reasonably available information is taken into consideration. Specifically, EPA will screen information developed after the completion of any IRIS assessment and evaluate the relevant information using OPPT's structured process . . . [60]

In the problem formulations themselves, however, EPA outlines a much broader approach. It indicates that *all* studies on IRIS-assessed chemicals will be reviewed using the "study quality" scoring system in EPA's TSCA systematic review document and other as-yet unidentified protocols for reviewing study relevance and weight.[61] This process would necessarily involve revisiting the interpretation of studies already evaluated in IRIS, potentially making different judgments about their quality and relevance and modifying overall IRIS determinations of the "best available science" and "weight of the evidence." Moreover, these judgments would be driven by a deeply flawed and unscientific method for reviewing studies that would result in less defensible conclusions than peer reviewed IRIS assessments.[62]

While TSCA section 26(h) establishes "scientific standards" for science-based decisions under section 6 and other provisions, these standards are general and flexible and do not materially change long-standing criteria used by agencies and the scientific community to assess the reliability, relevance and completeness of scientific evidence. The TSCA standards are consistent with the data review

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[60] EPA's Responses to Public Comments Received on the Scope Documents for the First Ten Chemicals for Risk Evaluation under TSCA, at 10.

[61] Typical is this description of EPA's approach in the problem formulation for asbestos, the subject of a comprehensive IRIS assessment:

> EPA expects to consider and analyze human health hazards as follows:
>
> 1) Included human health studies will be reviewed using the evaluation strategies laid out in the Application of Systematic Review in TSCA Risk Evaluations (U.S. EPA, 2018).
> - Studies will be evaluated using specific data evaluation criteria.
> - Study results will be extracted and presented in evidence tables by cancer endpoint.
>
> 2) Evaluate the weight of the scientific evidence of human health hazard data.
> - EPA will rely on the weight of the scientific evidence when evaluating and integrating human health hazard data. The data integration strategy will be designed to be fit-for-purpose in which EPA will use systematic review methods to assemble the relevant data, evaluate the data for quality and relevance, including strengths and limitations, followed by synthesis and integration of the evidence.
> - Assess dose-response information to refine quantitative unit risk for lung cancer and mesothelioma. Review the appropriate human data identified to update, or reaffirm, the 1988 quantitative estimate of the unit risk of asbestos-related lung cancer and mesothelioma by the inhalation route.
>
> 3) In evaluating reasonably available data, EPA will determine whether particular human receptor groups may have greater susceptibility to the chemical's hazard(s) than the general population.

Problem Formulation of the Risk Evaluation for Asbestos, at 51-52.

[62] See comments on the TSCA Systematic Review guidance from SCHF, NRDC, and UCSF-PRHE to Docket EPA-HQ-OPPT-2018-0210

methodologies used by IRIS, other EPA programs and expert organizations like NTP and provide no justification for questioning science judgments and study interpretations made in the IRIS process.

The drawbacks of reopening IRIS assessments are particularly troubling in the case of asbestos. The problem formulation indicates that EPA will review the asbestos database "with the goal of updating, or reaffirming, the unit risk." [63] It describes this review as follows:

> Asbestos has an existing EPA IRIS Assessment and an ATSDR Toxicological Profile; hence, many of the hazards of asbestos have been previously compiled and reviewed. EPA relied heavily on these comprehensive reviews in preparing the scope and problem formulation documents. EPA expects to use these documents as a starting point for identifying key and supporting studies to inform the human health hazard assessment, including dose-response analysis. EPA also expects to consider other studies that have been published since these reviews, as identified in the literature search conducted by the Agency for asbestos (Asbestos (CASRN 1332-21-4) Bibliography: Supplemental File for the TSCA Scope Document, EPA-HQ-OPPT-2016-0736). . . . The relevant studies will be evaluated using the data quality criteria in the Application of Systemic Review in TSCA Risk Evaluations document (U.S. EPA, 2018).[64]

There is no benefit – and considerable downside – in reconsidering the unit risk estimates provided by the IRIS program for asbestos of all fiber types (IRIS 1988) and Libby amphibole asbestos (IRIS 2014).[65] The highly flawed TSCA systematic review method for determining study "quality" would make it difficult for EPA to include important human health and toxicology studies in its chemical hazard assessments if there is any information that is missing or not publicly available. [66] Rejecting or downgrading epidemiological studies on asbestos on this ground could lead EPA to develop a new risk estimate that adopts the asbestos-industry position that chrysotile is safe – a position that was proposed by EPA under the George W. Bush Administration,[67] but rejected by the Scientific Advisory Board, which specifically warned that failure to consider epidemiology and toxicology data for asbestos is problematic.[68] These errors and scientific omissions could be repeated if application of the TSCA systematic review criteria results in discarding much of the asbestos epidemiology evidence.[69] This

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[63] Problem Formulation of the Risk Evaluation for Asbestos at 9.

[64] Id., at 34-35.

[65] IRIS 2014. Libby amphibole asbestos assessment. https://cfpub.epa.gov/ncea/iris2/chemicalLanding.cfm?substance_nmbr=1026

[66] See details documented in comments on the TSCA Systematic Review from SCHF, by NRDC, and by UCSF-PRHE to Docket EPA-HQ-OPPT-2018-0210

[67] EPA 2008. Proposed Approach for Estimation of Bin-Specific Cancer Potency Factors for Inhalation Exposure to Asbestos. https://www.epa.gov/sites/production/files/2015-11/documents/2008_prop_asbestos_approach.pdf

[68] SAB consultation on EPA's Proposed Approach for Estimation of Bin-Specific Cancer Potency Factors for Inhalation Exposure to Asbestos. Nov, 2008. EPA-SAB-09-004. https://yosemite.epa.gov/sab/sabproduct.nsf/77CFF6439C00ABF3852575010077801F/$File/EPA-SAB-09-004-unsigned.pdf

[69] See for example Table H-8 of the draft systematic review guidance which lists several pages of "serious flaws that would make epidemiological studies unacceptable for use," including failure to report various sorts of information, which is not considered a measure of study quality by any other peer reviewed systematic review framework.

would be a huge step back from the settled scientific consensus on the severe dangers of asbestos to public health.

Even without IRIS assessments, the risks of many substances have been thoroughly reviewed and determined by the Agency and other authoritative bodies but these earlier findings will now be subject to revision as EPA reinterprets studies using its TSCA systematic review document. For example, 1-Bromopropane is classified by the National Toxicology Program as "reasonably anticipated" to cause cancer in humans. In 2016 the EPA Draft Risk Assessment recognized the relevance and reliability of this health endpoint when it derived an inhalation unit risk estimate based on lung tumors. So, it is particularly disturbing that the problem formulation for this chemical states that the "the weight-of-evidence analysis for the cancer endpoint is inconclusive" and it will be evaluated using the flawed TSCA systematic review (EPA 2018 Problem Formulation, p. 45). The concern raised by SCHF, NRDC, and others regarding the industry bias of the TSCA systematic review document makes it likely that a re-analysis will result in a false negative – that is, discounting evidence of cancer (see comments on TSCA systematic review by SCHF, NRDC, Docket EPA-HQ-OPPT-2018-0210 incorporated by reference).

In sum, we strongly oppose any reopening of IRIS or other findings that have been finalized and represent authoritative determinations by the Agency. As it proceeds with the risk evaluations, EPA should rely on previous IRIS assessments except where significant new data are available. In this case, the IRIS program should evaluate whether the new data warrants modification of its previous determinations of the weight of the evidence for specific endpoints.

XI. EPA Risk Evaluations Should Not Reassess Uses of TCE, MC And NMP That Were Fully Assessed In Its Proposed Section 6(a) Rules for These Chemicals

EPA has proposed to ban certain uses of TCE, MC and NMP under section 6(a) of amended TSCA.[70] As the basis for these proposed rules, EPA conducted comprehensive exposure and risk assessments on the targeted uses of the three chemicals and concluded that these uses presented unreasonable risks of injury under TSCA. The EPA assessments were subject to public comment and peer review both during their development and again as part of the rulemaking process.

Although the EPA Administrator recently agreed to finalize the proposed MC ban, the problem formulations indicate that EPA will not rely on the completed assessments but will "reassess" the targeted uses for TCE and NMP.[71] We strongly disagree with this approach.

E.g., National Toxicology Program. Handbook for Conducting a Literature-Based Health Assessment Using OHAT Approach for Systematic Review and Evidence Integration. In: U.S. Department of Health and Human Services, editor.: Office of Health Assessment and Translation, Division of National Toxicology Program, National Institute of Environmental Health Sciences; 2015

[70] Trichloroethylene (TCE); Regulation of Use in Vapor Degreasing Under TSCA Section 6(a), 82 Fed. Reg. 7432 (Jan. 19, 2017); Trichloroethylene; Regulation of Certain Uses Under TSCA § 6(a), 81 Fed. Reg. 91592 (Dec. 16, 2016) and Methylene Chloride and N-Methylpyrrolidone; Regulation of Certain Uses Under TSCA Section 6(a), 82 Fed. Reg. 7464 (Jan. 19, 2017)

[71] See, e.g., Problem Formulation of the Risk Evaluation for Trichloroethylene, at 24-25.

In its peer reviewed IRIS assessment for TCE, EPA concluded that "[i]ncreased incidence of fetal cardiac malformations was identified as the most sensitive health endpoint within the developmental toxicity domain."[72] This finding was reaffirmed in EPA 2014 TCE Work Plan Chemical Assessment. In 2016, EPA scientists published a systematic review of the data confirming the basis for linking TCE exposure to congenital heart malformations.[73] Congenital heart effects can be disabling or even deadly. The significant and unreasonable risks posed by TCE in consumer and industrial products,[74] particularly from exposures during pregnancy, led EPA to propose to ban its use in aerosol and vapor degreasing operations.

Despite EPA's repeated findings of heart malformations linked to TCE, the problem formulation states that: "The relevant studies will be evaluated using the data quality criteria in the *Application of Systematic Review in TSCA Risk Evaluations document*."[75] This evaluation could result in EPA rejecting the peer-reviewed findings of earlier assessments. Significantly, at the same time as TSCA issued its systematic review guidance for public comment, an industry-sponsored consulting firm published its analysis of why the studies linking TCE with heart defects were "not sufficiently reliable for the development of toxicity reference values."[76] Since the industry-sponsored publication uses reasoning similar to that in the flawed TSCA systematic review guidance, it seems likely that the TSCA risk evaluation may similarly dismiss the evidence of congenital heart defects. Disregarding this important scientific evidence of harm would put the public at great risk.

It would be both scientifically indefensible and counterproductive for the Agency to reopen these assessments for yet another round of public input and to redo the extensive analyses they contain simply so industry commenters can have another bite at the apple on findings they dislike. The next step in the rulemakings should be to issue final rules as quickly as possible. These rules, once issued, should close the book on the targeted uses and enable EPA to focus its risk evaluations on uses that have not yet been assessed.

XII. EPA Should Not Presume That Occupational Exposure Standards Are Fully Protective of Workers, Can be Equated with the Absence of Unreasonable Risk and are Representative of Actual Worker Exposure

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[72] EPA 2018 TCE Problem Formulation, Section 2.4.2, page 45

[73] Makris SL, Scott CS, Fox J, Knudsen TB, Hotchkiss AK, Arzuaga X, Euling SY, Powers CM, Jinot J, Hogan KA, Abbott BD, Hunter ES 3rd, Narotsky MG. A systematic evaluation of the potential effects of trichloroethylene exposure on cardiac development. Reprod Toxicol. 2016 Oct;65:321-358.

[74] EPA 2017. Regulation of Certain Uses under Toxic Substances Control Act: Trichloroethylene. https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0163-0001
EPA 2017. Regulation of Certain Uses under Toxic Substances Control Act: Trichloroethylene; Vapor Degreasing. https://www.regulations.gov/document?D=EPA-HQ-OPPT-2016-0387-0001

[75] EPA 2018 TCE Problem Formulation, Section 2.4.2, page 44

[76] Wikoff D, Urban JD, Harvey S, Haws LC. Role of Risk of Bias in Systematic Review for Chemical Risk Assessment: A Case Study in Understanding the Relationship Between Congenital Heart Defects and Exposures to Trichloroethylene. Int J Toxicol. 2018 Mar/Apr;37(2):125-143.

Occupational exposure is significant for nearly all of the 10 chemicals and should be a major focus of EPA's risk evaluations. The problem formulations indicate that when evaluating occupational risks, the Agency will heavily weigh mandatory and voluntary workplace standards and "will consider the influence of the recommended exposure limits on occupational exposures."[77] We agree that existing workplace standards are relevant in determining risks to workers. However, for several reasons, it would be unjustified for EPA to presume that these standards are fully protective of workers or that their existence can be equated with the absence of unreasonable risk.

First, TSCA and the Occupational Safety and Health Act (OSH Act) apply differing standards of protection and the level of risk reduction afforded by OSHA limits may well be inadequate to satisfy the more stringent requirements of TSCA. OSHA is only authorized to adopt workplace standards for chemicals presenting "significant risks of harm," a term interpreted by the Supreme Court's *Benzene* decision as requiring OSHA to demonstrate by substantial evidence that "it is at least more likely than not that long-term exposure to [a chemical] presents a significant risk of material health impairment."[78] By contrast, the term "unreasonable risk" under TSCA does not impose this high threshold for regulation. Further, OSHA may impose only economically and technologically feasible limits on exposure.[79] However, economic and technological considerations have no bearing on EPA's determinations of unreasonable risk, which cannot take into account cost and other non-risk factors under section 6(b)(4)(A).[80] Finally, while OSHA is only authorized to place limits on exposure, TSCA provides a broad array of remedies, including bans of production and use, which may provide a level of protection that OSHA lacks authority to impose.

Second, a number of the OSHA standards that apply to chemicals subject to the first 10 risk evaluations were developed many years ago and do not reflect current data and scientific understanding of the health effects of the regulated chemicals.[81] Thus, the levels of exposure allowed by these standards may be unsafe when evaluated using the best available science.

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[77] See, e.g., Problem Formulation of the Risk Evaluation for 1-Bromopropane (May 2018), at 64.

[78] *Industrial Union Department, AFL-CIO v. American Petroleum Institute,* 448 U.S. 607 (1980)

[79] *American Textile Manufacturers Institute, Inc. v. Donovan*, 452 U.S. 490, 508-11 (1981).

[80] Based on these considerations, EPA decided against referring to OSHA workplace risks from exposure to trichloroethylene (TCE) under section 9(a) of TSCA, even though OSHA had earlier promulgated a workplace standard for TCE. In deciding to address risks to workers through a section 6(a) rulemaking instead, EPA compared its authority under TSCA to eliminate these risks to that of OSHA, concluding that "there is no other federal law that provides authority to prevent or sufficiently reduce these . . . exposures." It further concluded that risks that EPA found to be "unreasonable" under TSCA might not be deemed "significant" by OSHA. 82 Federal Register 7432, 7454 (January 19, 2017).

[81] OSHA has two types of standards. Under section 6(a) of the OSH Act, OSHA adopted hundreds of PELs in 1971 that were, at that time, considered national consensus standards. They have not been updated since and are based on science from the 1960s or earlier. Since 1971, OSHA has regulated only about 40 chemicals under section 6(b). These more comprehensive standards are based on thorough evaluation of health effects and a determination that risks are significant. OSHA has 6(b) standards regulating only asbestos and MC. It has PELs (adopted under 6(a)) for PERC and TCE but not for the other 10 chemicals. In the case of both asbestos and MC, OSHA's published Federal Register preambles found that even at the revised PEL, employees continued to be exposed to significant risks i.e., risks above 1/1000 – OSHA's definition of significant risk.

Third, OSHA does not cover all workers. It only covers private sector employees of employers. It does not cover employees of federal, state or local governments. These workers might include building maintenance people exposed to asbestos, hospital workers exposed to PERC when laundering linens or other supplies, etc. OSHA also does not cover independent contractors. In the construction sector, many people performing remodeling work, such as stripping paint and otherwise using MC, or removing asbestos insulation are independent. These workers have no OSHA protection. So even if OSHA standards were adequately protective of the workers they covered, there would still be a need for EPA to act under TSCA to make sure all workers had an equivalent level of protection.

Fourth, there is no basis for EPA to assume across-the-board compliance with OSHA standards. As the Agency pointed out in its proposed section 6(a) rule for MC paint removal products, exposures above the OSHA limit have been well documented.[82] To determine actual workplace exposures, we encourage EPA to obtain and review all the data gathered by law under OSHA's Access standard, 29 CFR 1910.1020 which "provide[s] employees and their designated representatives a right of access to relevant exposure and medical records; and to provide representatives of the Assistant Secretary a right of access to these records in order to fulfill responsibilities under the Occupational Safety and Health Act."[83] (1910.1020(a)). This would provide a basis for comparing actual exposures to OSHA standards and, for specific chemicals, determine whether and to what extent OSHA standards reliably limit exposure. While these data will provide a valuable snapshot of exposures, it should be kept in mind that OSHA exposure monitoring data is not systematic or comprehensive, and therefore may not be representative of workplace chronic or peak exposures that are likely to be missed with snapshot monitoring.

Finally, as EPA has recognized, some of the industrial hygiene strategies embodied in OSHA standards – such as labels and respirators – are known to be of limited effectiveness in protecting workers and have been required by OSHA to compensate for the lack of effective engineering controls or constraints on its authority, not because they are uniformly protective. For example, in its proposed section 6(a) rules for TCE, MC and NMP, EPA analyzed a universe of 48 studies[84] and concluded that:

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[82] Studies referenced by EPA found widespread non-compliance with the OSHA MC workplace standard during paint and coating removal, resulting in MC exposures above the OSHA standard, despite the mandatory nature of the OSHA requirements. 82 FR 7405 (Ref. 70)

[83] These data include:

- "Environmental (workplace) monitoring or measuring of a toxic substance or harmful physical agent, including personal, area, grab, wipe, or other form of sampling, as well as related collection and analytical methodologies, calculations, and other background data relevant to interpretation of the results obtained" (1910.1020(c)(5)(i)); and,
- "Biological monitoring results which directly assess the absorption of a toxic substance or harmful physical agent by body systems (e.g., the level of a chemical in the blood, urine, breath, hair, fingernails, etc.)" (excluding drug and alcohol testing) 1910.1020(c)(5)(ii).

For example, the OSHA standard for methylene chloride can be found at 29 CFR 1910.1052, which describes details of mandatory exposure monitoring, employee notification requirements, and long-term retention of the monitoring results. Under OSHA's Access standard, 29 CFR 1910.1020 (D)(7)(ii), employers must retain these records for 30 years.

[84] OPPT summarized these studies in a paper entitled:
The Effectiveness of Labeling on Hazardous Chemicals and Other Products (March 2016) (Ref. 33 in rulemaking docket).

> [C]onsumers and professionals do not consistently pay attention to labels; consumers and professional users often do not understand label information; consumers and professional users often base a decision to follow label information on previous experience and perceptions of risk; even if consumers and professional users have noticed, read, understood, and believed the information on a hazardous chemical product label, they may not be motivated to follow the label information, instructions, or warnings; and consumers and professional users have varying behavioral responses to warning labels, as shown by mixed results in studies.[85]

Similarly, EPA cautioned that "there are many documented limitations to successful implementation of respirators," explaining that:

> "Not all workers can wear respirators. Individuals with impaired lung function, due to asthma, emphysema, or chronic obstructive pulmonary disease for example, may be physically unable to wear a respirator. Determination of adequate fit and annual fit testing is required for a tight fitting full-face piece respirator to provide the required protection. Also, difficulties associated with selection, fit, and use often render them ineffective in actual application, preventing the assurance of consistent and reliable protection, regardless of the assigned capabilities of the respirator. Individuals who cannot get a good face piece fit, including those individuals whose beards or sideburns interfere with the face piece seal, would be unable to wear tight fitting respirators. In addition, respirators may also present communication problems, vision problems, worker fatigue and reduced work efficiency (63 FR 1156, January 8, 1998). According to OSHA, 'improperly selected respirators may afford no protection at all (for example, use of a dust mask against airborne vapors), may be so uncomfortable as to be intolerable to the wearer, or may hinder vision, communication, hearing, or movement and thus pose a risk to the wearer's safety or health. (63 FR 1189-1190).'"[86]

Because of these considerations, EPA cannot assume that, simply because they are required by OSHA standards, labeling or respirators will in fact provide adequate worker protection and successfully prevent unsafe exposure. Rather, as it did in its proposed rules for MC, TCE and NMP, EPA should explicitly recognize the limitations of these industrial hygiene controls and determine whether risks to workers are unreasonable given that labeling and respirators are often unprotective and unreliable in the real world.

Conclusion

The EPA problem formulations are replete with questionable exclusions and loopholes, failures to require necessary testing, deviations from accepted scientific methods and refusal to accept previous peer reviewed determinations of risk. As a result, the Agency is on a path to produce evaluations that ignore important exposure pathways and at-risk populations, disregard evidence of adverse effects and reach misleading, incomplete and understated conclusions about risk that weaken public health protection. EPA should put the 10 evaluations on hold, rethink how they are being conducted, and reinitiate them in accordance with the law and principles of sound science.

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[85] 81 FR at 91601.
[86] 82 FR 7445

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Michelle Naccarati-Chapkis
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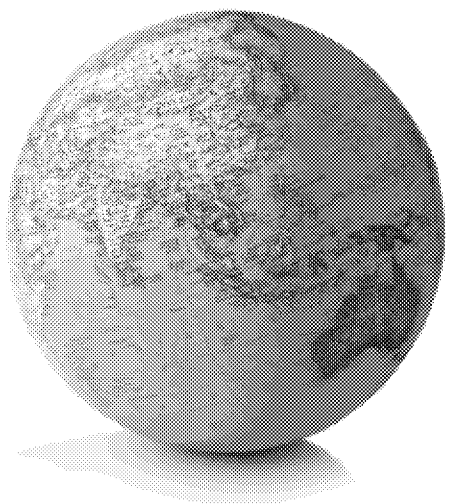
Women for a Healthy Environment

SQUIRE PATTON BOGGS

EPA's TSCA 6(a) Proposals: Regulation of Trichloroethylene (TCE) Use in Aerosol Degreasing, Use in Spot Cleaning by Dry Cleaners and Use in Vapor Degreasing



W. Caffey Norman
Squire Patton Boggs
March 10, 2017

Introduction

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- First rulemakings under TSCA § 6 in 27 years, will be first rulemakings interpreting "unreasonable risk" under revised TSCA.
- Three main points:
  - Failure to comply with Small Business Regulatory Enforcement Fairness Act (SBREFA)
  - Failure to comply with requirements of TSCA §§ 6, 26
  - Failure to comply with requirements of TSCA § 9
- In each case, the failure is clear from the face of the statute.

SBREFA Requirements

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- The Regulatory Flexibility Act, as amended by SBREFA, provides:
  - "**When any rule is promulgated which will have a significant economic impact on a substantial number of small entities**, the head of the agency promulgating the rule or the official of the agency with statutory responsibility for the promulgation of the rule shall assure that small entities have been given an opportunity to participate in the rulemaking. . . .
  - "**Prior to publication of an initial regulatory flexibility analysis which a covered agency is required to conduct by this chapter. . .the agency shall convene a review panel for such rule** consisting wholly of full time Federal employees of the office within the agency responsible for carrying out the proposed rule, the Office of Information and Regulatory Affairs within the Office of Management and Budget, and the Chief Counsel. . . .
  - "**If the head of the agency makes a certification**. . . **the agency shall publish such certification in the Federal Register at the time of publication of general notice of proposed rulemaking for the rule** or at the time of publication of the final rule, **along with a statement providing the factual basis for such certification**."

Noncompliance with SBREFA

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- No SBREFA Panel was held for the proposed spot cleaning/aerosol degreasing rule, however, as EPA certified that the rule would "not, if promulgated, have a significant economic impact on a substantial number of small entities."
  - Spot cleaning is done by dry cleaners, which are virtually all small entities.
  - It is not credible that EPA could certify that the rule would not have a significant economic impact on a substantial number of small entities (SISNOSE), where the National Cleaners Association (NCA) estimates that 60-90% of retail dry cleaners routinely use TCE on the spotting board (14,130 – 21,195 small businesses) and projects that such a ban will cost 4-5% of gross sales.
  - Even the lowest increased cost estimated by NCA (4% of gross sales), at the low end of the range of small dry cleaning entities (14,130), constitutes SISNOSE as defined in EPA's guidance (far more than the 1-3% impact on 100 small entities considered SISNOSE)..
  - Neither the preamble to the proposed rule nor the economic analysis contains the "statement providing the factual basis for such certification [of no SISNOSE]" required by SBREFA.

Failure to Give Notice

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- Draft TCE Work Plan assessment entitled "Degreaser and Arts/Crafts Uses"
- "EPA focused the assessment on uses of TCE as a degreaser (i.e., both in small commercial settings and by consumers or hobbyists) and on consumer use of TCE in products used by individuals in the arts and crafts field." (p. 14)
- Spot cleaning mentioned only in fn. 8: "there were several spot cleaners for fabrics marketed to consumers, but none contained TCE; lists of ingredients were not available for a few of the spot cleaners."
- No reference at all to spot cleaning in the workplace.
- With no explanation, final TCE Work Plan Assessment is entitled "Degreasing, *Spot Cleaning* and Arts & Crafts Uses" and includes "Commercial use of TCE as a spotting agent at dry cleaning facilities." (p. 26)
- Because there was no notice that EPA was addressing spot cleaning, there was no participation by dry cleaner representatives and no peer review of the spot cleaning assessment.

Failure to Comply with TSCA §§ 6, 26 Procedural Requirements

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- For risk assessments completed prior to passage of the Lautenberg Act, including that for TCE, TSCA § 26(l)(4) provides that "the Administrator may publish proposed and final rules under section 6(a) that are consistent with the scope of the completed risk assessment for the chemical substance and consistent with other applicable requirements of section 6."
- The failure to notify dry cleaners that EPA was assessing a key agent upon which they rely clearly violates TSCA § 6(b)(4)(H): "The Administrator shall provide no less than 30 days public notice and an opportunity for comment on a draft risk evaluation prior to publishing a final risk evaluation." This would appear to be an "applicable requirement[] of § 6" for purposes of TSCA § 26(l)(4), and in any event is required by § 553 of the Administrative Procedure Act.
- The supplemental analyses now in the docket for both proposals, done after enactment of the Lautenberg Act, also clearly require notice and peer review (discussed below).

Failure to Comply with TSCA §§ 6, 26 Scope Requirements

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- The proposed ban on TCE in vapor and aerosol degreasing also runs afoul of § 26(l)(4) because it is not "consistent with the scope of the completed risk assessment."
- The Work Plan Assessment is focused solely on exposure from TCE use as a solvent degreaser **in small commercial settings and by consumers:** "although the use of TCE as a solvent degreaser at large commercial/industrial operations is expected to be frequent and the concentration of TCE high, human exposures in these settings are expected to be monitored and controlled by Occupational Safety & Health Administration (OSHA); thus, this use is also not considered in this assessment" (p. 27).
- The proposed ban, however, recognizes no such limitation. EPA would prohibit all commercial use of TCE in vapor degreasing and general aerosol degreasing, whether by large or small operations.

Failure to Comply with TSCA §§ 6, 26 Science Requirements

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- Under TSCA § 6(b)(4)(F) the risk evaluation must:
  - "describe the weight of the scientific evidence for the identified hazard and exposure."
- TSCA § 26(h): "In carrying out sections 4, 5, and 6, to the extent that the Administrator makes a decision based on science, the Administrator shall use scientific information. . .employed in a manner consistent with the best available science. . . and shall consider as applicable—
  - (5) the extent of independent verification or peer review of the information. . . ."
- TSCA § 26(i): "The Administrator shall make decisions under sections 4, 5, and 6 based on the weight of the scientific evidence."

Failure to Comply with TSCA §§ 6, 26 Science Requirements, cont.

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- The Work Plan Assessment is inconsistent with applicable requirements of TSCA § 6 that it be based on weight of the evidence:
  - It expressly relies on hazard values derived directly from a single academic study to estimate non-cancer risk, even though three GLP-compliant studies conducted under EPA guidelines have been unable to reproduce the effect;
  - The University of Arizona study upon which EPA relies has been heavily criticized in the published literature, and other regulatory agencies have expressly declined to rely on the academic study citing data quality concerns;
  - The authors of the Arizona study have published repeated corrections that fail to address the data quality concerns; and a majority of EPA's own staff scientists expressed "low" confidence in its results;
  - It is a screening level assessment which does not meet OMB guidelines implementing the Information Quality Act for a "highly influential scientific assessment" to support TSCA § 6 rulemaking.

Failure to Comply with TSCA §§ 6, 26 Science Requirements, cont.

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- The Work Plan Assessment is inconsistent with applicable requirements of TSCA § 6 that it take into account peer review.
- EPA's peer review chairperson wrote: "The draft document fails to articulate satisfactorily that the analysis described within should be characterized as a screening level assessment. . . . I believe that the Agency acted prematurely in issuing this (screening level) assessment for public comment. . . . After listening carefully to the comments and contributions from the other members of the Panel, I have concluded that there would little benefit in revising this draft screening assessment."
- Another panelist devoted six pages to the non-cancer assessment alone: "fundamental biological problems with the OPPT non-cancer hazard indices and risk conclusions over concern for developmental toxicity render that aspect of the draft assessment unreliable. The draft document should be returned to the authors for major revision as there are substantial uncertainties in the domestic (hobby) exposure assessment and serious deficiencies in non-cancer hazard identification."

Failure to Comply with TSCA §§ 6, 26 Science Requirements, cont.

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- This highly unfavorable peer review was either ignored or, remarkably, characterized as favorable:
- Even though the BNA report of the review was entitled: *EPA Peer Reviewers Say Trichloroethylene Analysis Not Ready for Regulatory Use* (July 18, 2013), the EPA Assistant Administrator for Chemical Safety and Pollution Prevention wrote thereafter to the EPA Inspector General that "[i]t is notable **that the external peer reviews of all the Work Plan assessments we have completed thus far supported our overall assessment methodologies and conclusions**."

TSCA § 9

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- § 9(a) – Laws not administered by EPA:
  - If unreasonable risk can be sufficiently reduced under a law not administered by EPA, EPA shall publish and submit to the other agency a report and request it to determine if it can reduce the risk under such other law. The other agency must respond to EPA and publish its response.
  - Other agency must either decide that there is no such risk or initiate rulemaking within 90 days of its response
- § 9(b) – Laws administered by EPA:
  - If risk can be sufficiently reduced under another law administered by EPA, then EPA must use that other authority unless it determines that it is in the public interest to proceed under TSCA.
  - In making public interest determination, EPA must compare the estimated costs and efficiencies of the actions to be taken under TSCA and action to be taken under such other law.

Legislative History

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- Original history is clear: “it was the intent of the conferees that the Toxic Substance Act not be used, when another act is sufficient to regulate a particular risk.”
- Recent House report:“TSCA's original purpose [is] filling gaps in Federal law that otherwise did not protect against the unreasonable risks presented by chemicals,” and “the Administrator should respect the experience of, and defer to other agencies that have relevant responsibility such as the Department of Labor in cases involving occupational safety.” Colloquy:
- “Mrs. BLACKBURN. It is my understanding that, as a unified whole, this language, old and new, limits the EPA's ability to promulgate a rule under § 6 of TSCA to restrict or eliminate the use of a chemical when the Agency either already regulates that chemical through a different statute under its own control and that authority sufficiently protects against a risk of injury to human health or the environment, or a different agency already regulates that chemical in a manner that also sufficiently protects against the risk identified by EPA. Would the chairman please confirm my understanding of § 9?
- “Mr. SHIMKUS. The gentlewoman is correct in her understanding.
- “Mrs. BLACKBURN. As the EPA's early-stage efforts to regulate methylene chloride and TCE under TSCA § 6 illustrate, they are also timely. EPA simply has to account for why a new regulation for methylene chloride and TCE under TSCA is necessary. . . .”

Existing Workplace Regulation

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- OSHA regulates occupational exposure to TCE. The permissible exposure limits (PELs) are 100 ppm as an eight-hour TWA, 200 ppm as an acceptable ceiling concentration, and 300 ppm as an acceptable maximum peak (five minutes in any two-hour period) above the acceptable ceiling concentration for an eight-hour shift.
- TCE producers recommend compliance with TLVs developed by the American Conference of Governmental Industrial Hygienists. For TCE, the current TLVs are 10 ppm as an eight-hour TWA and 25 ppm as a Short-Term Exposure Limit.

Existing Environmental Regulation -- Vapor Degreasing

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- Halogenated Solvent Cleaning NESHAP, 40 C.F.R. Part 63, Subpart T
  - 59 Fed. Reg. 61800 (Dec. 2, 1994) – MACT for major and area sources
  - 72 Fed. Reg. 25138 (May 3, 2007) – 14,100 kg/year facility-wide emissions limit ("ample margin of safety to protect public health")
- Changed work practices, reduced in-facility exposure (occupational and bystander) and fenceline emissions
- Work Plan Assessment relies on data collected before the May 2010 compliance deadline for NESHAP (primarily the NEI and TRI, and many assumptions (see pp. 34-37)) to estimate releases, exposures, and population exposed (pp. 114-15). This major source of uncertainty could be eliminated by reference to data required to be reported under the NESHAP:
  - Under the NESHAP, every facility must make initial notification and report annually to EPA for each degreaser: type of machine and controls, location, date of installation, solvent consumption, and emissions.

Existing Environmental Regulation -- Vapor Degreasing, cont.

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- More basically, to extent Work Plan Assessment references NESHAP at all, it reflects misunderstanding of it:
  - "EPA's overall emission limit for implementing [the NESHAP] is 150 kilograms (kg) per square meter (m^2) per month (EPA, 2004a)" (p. 39).
  - This reference is to NESHAP for organic liquids distribution (non-gasoline), not here relevant.
  - 150 kg/m^2 per month limit was an alternative standard for batch machines in 1994 degreasing NESHAP (MACT).
- Current emissions limit is 14,100 kg/year facility-wide TCE emissions; not reflected at all in Assessment.

Failure to Comply with TSCA § 9

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- EPA refers only to consultation with and letter from OSHA to support taking over regulation of toxic substances in workplace from OSHA. No report, no referral, no publication, no apparent effort to comply with § 9(a).
- Erroneous references to EPA's own NESHAP for vapor degreasing and failure to use data collected under NESHAP to inform exposure estimates undermine determination under § 9(b) that it is in the public interest to proceed under TSCA. Also no required comparison of the estimated costs and efficiencies of the actions to be taken under TSCA and actions taken under other law.
- Enormous precedential impact.

Vapor Degreasing -- Small Business Cost Impacts

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- Precision Machined Products Association (240 shops) commented at SBAR:

  - Cost to replace existing degreasing equipment ranges from $350,000-500,000, workplace modifications often required to accommodate larger systems.
  - Cost estimates range from 25% of net revenue to total annual profit for some of the shops consulted.
  - Expenditure of $350,000-500,000 is equivalent to 2-3 years of planned capital investments and would leave US shops far behind South Asian competitors.
  - Such expenditure would starve PMPA members of capital to upgrade their current processes, purchase new equipment, and make needed improvements.
  - Smaller companies (12-75 employees) report that a mandate to replace cleaning equipment requiring $350,000 or more would be a tipping point decision regarding closing or maintaining the business.
  - Shop closings would put all employees out of work and destroy millions in owner's equity as the business assets would be liquidated.
  - One shop said that $500,000 cost for new cleaning technology would consume its total planned 5-year capital investment budget.

Vapor Degreasing -- Small Business Technical Impacts

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- Where TCE is used it is generally sole means of parts cleaning -100% of shop output.
- Shops investigating alternatives found no comparable cleanliness except using NPB, which is not a viable alternative as it is toxic for worker exposure above TLV (0.1 ppm).
- Aerospace, defense, medical and automotive contracts lock in cleaning methods as part of the approval process. Customers typically demand that critical machined parts be free from oil.
- Failure to remove oil completely can affect reliability of automatic optical and electronic gauging systems in place to assure 100% verification on safety-critical automotive and aerospace parts.
- Compatibility of replacement cleaners an issue as TCE is accepted for compatibility with polymers, especially important in defense applications (*e.g.*, many shops in Europe have received derogations due to requirements of BAE, Airbus, and others for parts to be cleaned with TCE).
- One shop making airbag, braking, and other engine mount parts estimated that approval of a new cleaning process by automotive customers would entail 5-10 man-years to test, document results, prepare automotive FMEA/PPAP documentation, submit, and follow up with customers for approval.

BEFORE THE ENVIRONMENTAL PROTECTION AGENCY

Trichloroethylene; Regulation of Certain Uses under TSCA § 6(a)

[EPA-HQ-OPPT-2016-0163; FRL-9949-86]

Comments of the

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March 16, 2017

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The Halogenated Solvents Industry Alliance, Inc. (HSIA) represents producers and users of trichloroethylene (TCE). We offer these comments on EPA's proposed rule banning manufacture of TCE for and use of TCE in aerosol degreasing and in spot cleaning by dry cleaning facilities. 81 Fed. Reg. 91592 (Dec. 16, 2016). This rule, proposed under § 6(a) of the Toxic Substances Control Act (TSCA), is based on a Work Plan Assessment of TCE completed by EPA in June 2014. TSCA was amended in June 2016 by the Frank R. Lautenberg Chemical Safety for the 21st Century Act ("Lautenberg Act").

HSIA urges EPA to withdraw the proposed rule, which is based on a very deficient risk assessment.. While EPA is authorized under TSCA § 26(l)(4) to propose a § 6 rule based on a risk assessment completed before TSCA was revised, there is no requirement or deadline for it to do so. The situation is very different for the ten priority compounds designated by EPA under TSCA § 6(b)(2)(A) in December 2016.[1] For these ten designated pollutants, TSCA establishes deadlines for risk assessments and rulemakings. TCE is one of the ten priority compounds, and the better course would be to assess the risks from spot cleaning and aerosol degreasing as part of the required upcoming TCE assessment.

These comments address the following subjects, among others, in detail:

- TSCA § 26(l)(4) requires, for a rule based on a risk assessment completed before TSCA was revised, that the rule must be consistent with "the scope of the completed risk assessment for the chemical substance and consistent with other applicable requirements of § 6." "Although the use of TCE as a solvent degreaser at large commercial/industrial operations" was "not considered in this assessment," EPA nevertheless would prohibit all "commercial use of TCE in aerosol degreasing products," regardless of the size of the facility. This is plainly outside "the scope of the completed risk assessment."

- Further, the TCE Work Plan Assessment does not comply with the requirements of TSCA § 6(b)(4)(F) or TSCA § 26(h) and (i), which are expressly applicable to any EPA "decision based on science" under TSCA § 6. The disparity between the completed risk assessments and the "applicable requirements of § 6" is obvious from even a cursory review of the procedures for risk evaluation under the amended TSCA proposed by EPA earlier this year.

- The Work Plan Assessment expressly relied on hazard values derived directly from a University of Arizona study to estimate non-cancer risk. Several other studies, including two GLP-compliant studies conducted under EPA and OECD guidelines, have been unable to reproduce the effect seen in the Arizona study. The Arizona study has been heavily criticized in the published literature, its results have not been replicated by any other laboratory, and other regulatory authorities (including the California EPA) have rejected the study as deficient.

- Equally, the Work Plan Assessment relies on qualitative and quantitative estimates of cancer risk that are not realistic or justified by any underlying science. EPA estimates a baseline cancer risk from chronic occupational spot cleaning exposures of 1 in 10. Cancer incidence of this magnitude could not go unnoticed, and indeed EPA's estimate is belied by available epidemiology studies of dry cleaning workers which show no such risk. Indeed, two recent large Nordic epidemiological studies, both of which had extensive follow-up of the cohorts, have failed to find an association between TCE and kidney cancer, and these are not addressed in the Work Plan Assessment. Further, EPA's development of a potency factor based on Charbotel *et al.* (2006) directly contravenes the advice EPA received from the National Academy of Sciences.

[1] 81 Fed. Reg. 91927 (Dec.19, 2016).

- On the exposure side, for spot cleaning EPA relied solely on a 2007 California study, which it recognized may not be representative of US dry cleaning facilities. For aerosol degreasing EPA provided no emissions or monitoring data – thus these are hypothetical exposures. Moreover, the draft TCE assessment, entitled "Degreaser and Arts/Crafts Uses," did not address spot cleaning (except to say that none of those sold to consumers contained TCE), but the final Work Plan Assessment is entitled "Degreasing, *Spot Cleaning* and Arts & Crafts Uses" and includes commercial use of TCE as a spotting agent at dry cleaning facilities.

- Because there was no notice that EPA was addressing spot cleaning, there was no participation by dry cleaner representatives and no peer review of the spot cleaning assessment. Moreover, there was no Small Business Advocacy Review, even though spot cleaning is done by dry cleaners which are virtually all small entities. It is not credible that EPA could certify that the rule would not have a significant economic impact on a substantial number of small entities (SISNOSE), where the dry cleaning industry estimates that 60-90% of retail dry cleaners routinely use TCE on the spotting board (14,130 – 21,195 small businesses) and projects that such a ban will cost 4-5% of gross sales, far more than the 1-3% impact considered SISNOSE.

- Peer review of the draft Work Plan Assessment was scathing. Reliance on the unreproducible Arizona study was harshly criticized. The Chair of the panel noted that it was a screening level assessment, not suitable for use in regulation: "*the Agency acted prematurely in issuing this (screening level)* assessment for public comment. . . . After listening carefully to the comments and contributions from the other members of the Panel, I have concluded that there would *little benefit in revising this draft screening assessment.*"[2] Yet EPA claims the peer review was supportive.

- EPA's determination that TCE use in spot cleaning and aerosol degreasing poses an "unreasonable risk" is based on its assessment of risks to workers. It is clear, however, that TSCA is to be used only when other statutes fail to provide a remedy for unreasonable risks. Worker health and safety fall under the jurisdiction of the Occupational Safety and Health Administration (OSHA), and use of TCE in spot cleaning and spray degreasing is already adequately regulated under the Occupational Safety and Health Act. Congress cannot have meant, in enacting "gap-filling" legislation, to open the door to EPA assuming all authority over the use of hazardous substances in the workplace.

I. Failure of Work Plan Assessment to Comply with TSCA §§ 6, 26

A. Applicable Requirements of TSCA §§ 6, 26

Although the Lautenberg Act made significant changes to TSCA to ensure that EPA would employ the "best available science" in its risk assessments, EPA proposes to rely on a remarkably sketchy and inadequate assessment in its inaugural rulemaking under TSCA § 6. TSCA § 6(b)(4)(F), as revised by the Lautenberg Act, requires that EPA's risk evaluations must, among other things:

- "integrate and assess available information on hazards and exposures for the conditions of use of the chemical substance, including information that is relevant to specific risks of injury to health or the environment and information on potentially exposed or susceptible subpopulations identified as relevant by the Administrator;"

[2] https://www.epa.gov/sites/production/files/2015-09/documents/tce_consolidated_peer_review_comments_september_5_2013.pdf.

- "take into account, where relevant, the likely duration, intensity, frequency, and number of exposures under the conditions of use of the chemical substance;" and

- "describe the weight of the scientific evidence for the identified hazard and exposure."

New TSCA § 26(h) requires that, in carrying out § 6, "to the extent that the Administrator makes a decision based on science, the Administrator shall use scientific information, technical procedures, measures, methods, protocols, methodologies, or models, employed in a manner consistent with the best available science, and shall consider as applicable—

> (1) the extent to which the scientific information, technical procedures, measures, methods, protocols, methodologies, or models employed to generate the information are reasonable for and consistent with the intended use of the information;
>
> (2) the extent to which the information is relevant for the Administrator's use in making a decision about a chemical substance or mixture;
>
> (3) the degree of clarity and completeness with which the data, assumptions, methods, quality assurance, and analyses employed to generate the information are documented;
>
> (4) the extent to which the variability and uncertainty in the information, or in the procedures, measures, methods, protocols, methodologies, or models, are evaluated and characterized; and
>
> (5) the extent of independent verification or peer review of the information or of the procedures, measures, methods, protocols, methodologies, or models."

With regard to risk assessments completed prior to passage of the Lautenberg Act, including that for TCE, TSCA § 26(l)(4) provides that "the Administrator may publish proposed and final rules under section 6(a) that are consistent with the scope of the completed risk assessment for the chemical substance and consistent with other applicable requirements of section 6." Thus, EPA may base regulation on the pre-enactment risk assessments only to the extent that they comply with the substantive requirements above.

Regrettably, the proposal to ban TCE in aerosol degreasing addresses a broader scope of uses than considered in the Work Plan Assessment. The scope of that assessment is clear: "although the use of TCE as a solvent degreaser at large commercial/industrial operations is expected to be frequent and the concentration of TCE high, human exposures in these settings are expected to be monitored and controlled by Occupational Safety & Health Administration (OSHA); thus, this use is also not considered in this assessment" (p. 27). The Assessment is focused solely on exposure from TCE use as a solvent degreaser in small commercial settings and by consumers.[3] The proposed ban, however, recognizes no such limitation. It would prohibit commercial use of TCE for general aerosol degreasing, as well as its manufacture, processing, and distribution in commerce for this use. Because the proposed rule would ban uses beyond the scope of the underlying Work Plan Assessment, it is not "consistent with the scope of the completed risk assessment" and therefore does not comply with TSCA § 26(l)(4).

[3] *See* Work Plan Assessment at Table 1-1.

Further, the proposed rule does not comply with the requirements of TSCA § 6(b)(4)(F) or TSCA § 26(h) and (i), which are expressly applicable to any EPA "decision based on science" under TSCA § 6. The disparity between the completed TCE Work Plan Assessment and the "applicable requirements of § 6" is obvious from a review of the procedures for risk evaluation under the amended TSCA proposed by EPA earlier this year.[4]

B. Deficiencies of Principal Non-Cancer Study

1. Not Reproducible

The Work Plan Assessment expressly relies on hazard values derived directly from a single academic study to estimate non-cancer risk.[5] Specifically, it states (p. 104):

> "The acute inhalation risk assessment used developmental toxicity data to evaluate the acute risks for the TSCA TCE use scenarios. As indicated previously, EPA's policy supports the use of developmental studies to evaluate the risks of acute exposures. This policy is based on the presumption that a single exposure of a chemical at a critical window of fetal development, as in the case of cardiac development, may produce adverse developmental effects (EPA, 1991).
>
> "After evaluating the developmental toxicity literature of TCE, the TCE IRIS assessment concluded that the fetal heart malformations are the most sensitive developmental toxicity endpoint associated with TCE exposure (EPA, 2011e). Thus, EPA/OPPT based its acute risk assessment on the most health protective endpoint (i.e., fetal cardiac malformations; Johnson et al., 2003) representing the most sensitive human population (i.e., adult women of childbearing age and fetus >16 yrs).
>
> "The acute risk assessment used the PBPK-derived hazard values (HEC_{50}, HEC_{95}, or HEC_{99}) from Johnson et al. (2003) developmental study for each degreaser and spot cleaner use scenario. . . . These extremely low values result in margin of exposure ("MOE") values below 10 for almost all the occupational and residential exposure scenarios examined."

A single flawed study should not be the basis for the toxicological value that serves as the basis for regulation. Several other studies, including three GLP-compliant studies conducted under EPA guidelines to support pesticide registration (40 CFR § 870.3700) and Organization for Economic Coordination & Development ("OECD") guidelines (414) have been unable to reproduce the effect seen by Johnson *et al.* (2003).

Johnson *et al.* (2003) reported cardiac effects in rats from research carried out at the University of Arizona and originally published ten years earlier by the same authors.[6] In the earlier-published study, there was no difference in the percentage of cardiac abnormalities in rats dosed during both pre-mating

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[4] 82 Fed. Reg. 7562 (Jan. 19, 2017).

[5] Johnson PD, *et al.*, Threshold of trichloroethylene contamination in maternal drinking waters affecting fetal heart development in the rat, Environ Health Perspect. 111:289-92 (2003).

[6] Dawson, B, *et al.*, Cardiac teratogenesis of halogenated hydrocarbon-contaminated drinking water, J. Am. Coll. Cardiol. 21: 1466-72 (1993).

and pregnancy at drinking water exposures of 1100 ppm (9.2%) and 1.5 ppm (8.2%), even though there was a 733-fold difference in the concentrations. The authors reported that the effects seen at these exposures were statistically higher than the percent abnormalities in controls (3%). For animals dosed only during the pregnancy period, the abnormalities in rats dosed at 1100 ppm (10.4%) were statistically higher than at 1.5 ppm (5.5%), but those dosed at 1.5 ppm were not statistically different from the controls. Thus, no meaningful dose-response relationship was observed in either treatment group. Johnson *et al.* republished in 2003 data from the 1.5 and 1100 ppm dose groups published by Dawson *et al.* in 1993 and pooled control data from other studies, an inappropriate statistical practice, to conclude that rats exposed to levels of TCE greater than 250 ppb during pregnancy have increased incidences of cardiac malformations in their fetuses.

2. Criticism in Literature and by Other Regulators

Johnson *et al.* (2003) has been heavily criticized in the published literature.[7] Indeed, its predecessor study was expressly rejected as the basis for MRLs by the Agency for Toxic Substances & Disease Registry (ATSDR) in its last TCE Toxicological Profile Update.[8] Moreover, the Johnson *et al.* (2003) findings were not reproduced in a study designed to detect cardiac malformations; this despite employing an improved method for assessing cardiac defects and the participation of Dr. Johnson herself.[9] No increase in cardiac malformations was observed in the second guideline study,[10] despite high inhalation doses and techniques capable of detecting most of the malformation types reported by Johnson *et al.* (2003). The dose-response relationship reported in Johnson *et al.* (2003) for doses spanning an extreme range of experimental dose levels is considered by many to be improbable, and has not been replicated by any other laboratory.[11]

Even the California Office of Environmental Health Hazard Assessment (OEHHA) rejected the study as deficient:

> "Johnson et al. (2003) reported a dose-related increased incidence of abnormal hearts in offspring of Sprague Dawley rats treated during pregnancy with 0, 2.5 ppb, 250 ppb, 1.5 ppm, and 1,100 ppm TCE in drinking water (0, 0.00045, 0.048, 0.218, and 128.52 mg/kg-day, respectively). The NOAEL for the Johnson study was reported to be 2.5 ppb (0.00045 mg/kg-day) in this short exposure (22 days) study. The percentage of abnormal hearts in the control group was 2.2 percent, and in the treated groups was 0 percent (low dose), 4.5 percent (mid dose 1), 5.0 percent (mid dose 2), and 10.5 percent (high dose).

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[7] Hardin, B, *et al.*, Trichloroethylene and cardiac malformations, Environ. Health Perspect. 112: A607-8 (2004); Watson, R., *et al.*, Trichloroethylene-contaminated drinking water and congenital heart defects: a critical analysis of the literature, Repro. Toxicol. 21: 117-47 (2006).

[8] ATSDR concluded that "[t]he study is limited in that only two widely spaced exposure concentrations were used and that a significant dose-response was not observed for several exposure scenarios." Toxicological Profile for Trichloroethylene Update (September 1997), at 88.

[9] Fisher, J, *et al.*, Trichloroethylene, trichloroacetic acid, and dichloroacetic acid: do they affect fetal rat heart development? Int. J. Toxicol. 20: 257-67 (2001).

[10] Carney, E, *et al.*, Developmental toxicity studies in Crl:Cd (SD) rats following inhalation exposure to trichloroethylene and perchloroethylene, Birth Defects Research (Part B) 77: 405–412 (2006).

[11] "Johnson and Dawson, with their collaborators, are alone in reporting that TCE is a 'specific' cardiac teratogen." Hardin, B, *et al.*, Trichloroethylene and cardiac malformations, Environ. Health Perspect. 112: A607-8 (2004).

> The number of litters with fetuses with abnormal hearts was 16.4 percent, 0 percent, 44 percent, 38 percent, and 67 percent for the control, low, mid 1, mid 2, and high dose, respectively. The reported NOAEL is separated by 100-fold from the next higher dose level. The data for this study were not used to calculate a public-health protective concentration since a meaningful or interpretable dose-response relationship was not observed. *These results are also not consistent with earlier developmental and reproductive toxicological studies done outside this lab in mice, rats, and rabbits: The other studies did not find adverse effects on fertility or embryonic development, aside from those associated with maternal toxicity (Hardin et al., 2004).*"[12]

3. Reservations of EPA Scientific Staff

Remarkably, an EPA staff review that was placed in the docket for the Work Plan Assessment reflects similar concerns. First, one staff member dissented over relying at all on the Arizona study:

> "The rodent developmental toxicology studies conducted by Dawson et al. (1993), Johnson et al. (2003), and Johnson et al. (1998) that have reported cardiac defects resulting from TCE (and metabolite) drinking water exposures have study design and reporting limitations. Additionally, two good quality (GLP) inhalation and gavage rodent studies conducted in other laboratories, Carney et al. (2006) and Fisher et al. (2001), respectively, have not detected cardiac defects. These limitations and uncertainties were the basis of the single dissenting opinion of a team member regarding whether the database supports a conclusion that TCE exposures during development are likely to cause cardiac defects."[13]

Second, even the EPA staff that agreed with use of the study had little confidence that it supported the dose-response assessment:

> "[A] majority of the team members agreed that the Johnson et al. (2003) study was suitable for use in deriving a point of departure. However, confidence of team members in the dose response evaluation of the cardiac defect data from the Johnson et al. (2003) study was characterized as between 'low' and 'medium' (with 7 of 11 team members rating confidence as 'low' and four team members rating confidence as 'low to medium')."[14]

It is surprising that EPA would consider use of a dose-response value for regulation from a study in which seven of its own scientists expressed "low" confidence, and in which the other four could muster no more than "low to medium" confidence. The same report notes: "In conclusion, there has not been a confirmation of the results of the Johnson et al. (2003) and Dawson et al. (1993) studies by another laboratory, but there has also not been a repeat of the exact same study design that would corroborate or refute their findings."

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[12] California EPA Public Health Goal for Trichloroethylene in Drinking Water (July 2009), at 21 (emphasis added).

[13] TCE Developmental Cardiac Toxicity Assessment Update (available at http://www.regulations.gov/#!documentDetail;D=EPA-HQ-OPPT-2012-0723-0045).

[14] *Id.*

4. EPA's Dose-Response Evaluation using Johnson *et al.* (2003) Is Inappropriate

The TCE Work Plan Assessment relies on the prior IRIS Assessment's evaluation of the relationship between TCE exposure dose and the development of cardiac defects, as described in Johnson *et al.* (2003). Ignoring for the moment the myriad of methodological deficiencies in the paper, a closer look at EPA's evaluation of that dose-response relationship in generating a point of departure (POD) raises several concerns. The importance of this activity cannot be overstated, as according to a paper published by the authors of the IRIS Assessment, Johnson *et al.* (2003) represents "the only available study potentially useable for dose-response analysis of fetal cardiac defects."[15]

In discussing the dose-response evaluation, Makris *et al.* (2016) further state that "[g]iven the uncertainties in the dose-response analysis related to the nature of the data, the confidence in the POD based on Johnson *et al.* (2003) has limitations. Overall, however, the POD derived in the 2011 TCE assessment (U.S. EPA, 2011), which used an approach consistent with standard U.S. EPA dose-response practices, remains a reasonable choice." It should be noted that, in order to achieve a better model fit in its derivation of a POD, EPA dropped the highest exposure dose from Johnson *et al.* (2003). With already questionable data, and no expectation that the highest dose of TCE would result in a diminished response, that decision should be reconsidered.

Makris *et al.* (2016) describe additional dose-response analyses performed to characterize the uncertainty in the POD. In summarizing the results of this analysis, they state that "[a]lternative PODs were derived based on use of alternative models, alternative BMR levels, or alternative procedures (such as LOAEL/NOAEL approach), each with different strengths and limitations. These alternatives were within *about an order of magnitude of the POD derived in the 2011 TCE assessment*" (emphasis added). This level of uncertainty in modeling the POD when combined with the uncertainty in the PBPK modeling (discussed elsewhere) and the overall poor quality of the underlying developmental toxicity study provide little confidence in the resulting non-cancer toxicological value in the Work Plan Assessment that drives the proposed regulation.

5. Reliance on Johnson *et al.* (2003) Is Inconsistent with Use of Best Available Science

All acute inhalation exposures in the TCE Work Plan Assessment were measured against potential developmental toxicity endpoints based solely on EPA's IRIS evaluation of Johnson *et al.* (2003). When HSIA requested access to the data used by EPA in its evaluation of the dose-response relationship between TCE exposure and cardiac defects reported in Johnson *et al.* (2003), the Agency provided the spreadsheet, referenced as Johnson (2009) (HERO ID 783484) in the 2011 IRIS Assessment, and indicated that was the entirety of the data evaluated. Examination of that spreadsheet reveals an absence of certain critical information, including, most importantly, dates for any of the individual treatment/control animals.

Acknowledging the documented deficiencies in their paper (and the data provided to EPA), the authors published an erratum aimed at updating the public record regarding methodological issues for Johnson *et al.* (2003).[16] According to Makris *et al.* (2016):

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[15] Makris SL, Scott CS, Fox J, *et al.*, Systematic evaluation of the potential effects of trichloroethylene exposure on cardiac development. Repro Toxicol (2016); http://dx.doi.org/10.1016/j.reprotox.2016.08.014

[16] Johnson PD, Goldberg SJ, Mays MZ, Dawson BV, Erratum: Erratum for Johnson *et al.* [Environ Health Perspect 113: A18 (2005)]; Environ Health Perspect 122: A94 (2014); http://dx.doi.org/10.1289/ehp.122-A94

"some study reporting and methodological details remain unknown, *e.g.*, the precise dates that each individual control animal was on study, maternal body weight/food consumption and clinical observation data, and the detailed results of analytical chemistry testing for dose concentration. Additional possible sources of uncertainty identified for these studies include that the research was conducted over a 6-yr period, that combined control data were used for comparison to treated groups, and that exposure characterization may be imprecise because tap (rather than distilled) drinking water was used in the Dawson *et al.* (1993) study and because TCE intake values were derived from water consumption measures of group-housed animals."

HSIA submits that the information contained in the above paragraph alone should disqualify Johnson *et al.* (2003) as "best available science" as required under EPA's proposed procedures for chemical risk evaluation under TSCA as amended.[17]

6. Failure to Conform to EPA Guidelines for Developmental Toxicity Risk Assessment

EPA's Guidelines for Developmental Toxicity Risk Assessment establish the framework for evaluation of developmental toxicity risk on a case-by-case basis.[18] Under these Guidelines, "[i]f data are considered *sufficient* for risk assessment, an oral or dermal reference dose for developmental toxicity (RfD_{DT}) or an inhalation reference concentration for developmental toxicity (RfC_{DT}) is then derived for comparison with human exposure estimates" (emphasis added).

In defining sufficiency, the Guidelines state: "In the case of animal data, agents that have been tested adequately in laboratory animals *according to current test guidelines* generally would be included in the "Sufficient Experimental Animal Evidence/Limited Human Data" category (emphasis added)." Where, as here, the "database on a particular agent includes less than the minimum sufficient evidence (as defined in the 'Insufficient Evidence' category) necessary for a risk assessment, but some data are available, this information could be used to determine the need for additional testing. . . . In some cases, a database may contain conflicting data. In these instances, the risk assessor must consider each study's strengths and weaknesses within the context of the overall database in an attempt to define the strength of evidence of the database for assessing the potential for developmental toxicity."

Given the demonstrated shortcomings of Johnson *et al.* (2003), which was not conducted to EPA test guidelines, and the availability to EPA of three guideline studies, we submit that the Guidelines for Developmental Toxicity Risk Assessment and TSCA §§ 6 and 26 require a weight of evidence evaluation of the database before EPA relies on Johnson *et al.* (2003) for regulatory purposes.

7. New Relevant Information

A third guideline study of TCE developmental toxicity has been sponsored by HSIA. The study was designed with a focus on cardiac abnormalities and included toxicokinetic measures to enable comparison with the earlier studies. It was intended to fill the remaining gap for a guideline study by the drinking water route, the same exposure route as Johnson *et al.* (2003). Regrettably, although the in-life

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[17] 82 Fed. Reg. 7562 (Jan. 19, 2017).

[18] 56 Fed. Reg. 63798 (December 5, 1991).

portion of the study was conducted during October and November, 2016, the concentrations of TCE measured in the drinking water solutions were found to be below the acceptable target range of 100% ± 10%, invalidating the study. The laboratory is conducting additional studies to identify the source of the deviations and the study will be rerun as soon as the dosing methodological issues are resolved and scheduling permits. A statement to this effect is attached as Appendix 1.

C. Deficiencies of Cancer Risk Assessment

1. Erroneous Characterization of TCE as "Carcinogenic to Humans"

While acute risks of developmental toxicity are characterized by EPA as of the greatest concern, the Work Plan Assessment also concludes that all but one of the degreaser exposure scenarios exceeded all the target cancer levels. The discussion of carcinogenicity in the Work Plan Assessment suffers from slavish reliance on EPA's earlier assessment of TCE under its Integrated Risk Information System.[19] The IRIS Assessment classifies TCE as "Carcinogenic to Humans." It fails to discuss (or even to recognize) that such classification is inconsistent with a definitive report by the National Academy of Sciences, discussed below.[20] We briefly address below how the epidemiological data on TCE do not meet the threshold for classification as "Carcinogenic to Humans."

a. Guidelines for Carcinogen Risk Assessment

EPA's 2005 Guidelines for Carcinogen Risk Assessment provide the following descriptors as to the weight of evidence for carcinogenicity:

- Carcinogenic to humans,
- Likely to be carcinogenic to humans,
- Suggestive evidence of carcinogenicity,
- Inadequate information to assess carcinogenic potential, and
- Not likely to be carcinogenic to humans.[21]

According to the Guidelines, "carcinogenic to humans" means the following:

"This descriptor indicates strong evidence of human carcinogenicity. It covers different combinations of evidence.

- "This descriptor is appropriate when there is convincing epidemiologic evidence of a causal association between human exposure and cancer.

[19] "Toxicological Review of Trichloroethylene (CAS No. 79-01-6) in Support of Summary Information on the Integrated Risk Information System (IRIS)" ("IRIS Assessment")

[20] National Research Council, Contaminated Water Supplies at Camp Lejeune: Assessing Potential Health Effects (2009) (hereinafter "Camp Lejeune report").

[21] 70 Fed. Reg. 17766-817 (April 7, 2005).

- "Exceptionally, this descriptor may be equally appropriate with a lesser weight of epidemiologic evidence that is strengthened by other lines of evidence. It can be used when *all* of the following conditions are met: (a) There is strong evidence of an association between human exposure and either cancer or the key precursor events of the agent's mode of action but not enough for a causal association, *and* (b) there is extensive evidence of carcinogenicity in animals, *and* (c) the mode(s) of carcinogenic action and associated key precursor events have been identified in animals, *and* (d) there is strong evidence that the key precursor events that precede the cancer response in animals are anticipated to occur in humans and progress to tumors, based on available biological information. In this case, the narrative includes a summary of both the experimental and epidemiologic information on mode of action and also an indication of the relative weight that each source of information carries, *e.g.*, based on human information, based on limited human and extensive animal experiments."

According to the Guidelines, the descriptor "likely to be carcinogenic to humans":

"is appropriate when the weight of the evidence is adequate to demonstrate carcinogenic potential to humans but does not reach the weight of evidence for the descriptor 'Carcinogenic to Humans.' Adequate evidence consistent with this descriptor covers a broad spectrum. . . . Supporting data for this descriptor may include:

"An agent demonstrating a plausible (but not definitively causal) association between human exposure and cancer:

- "An agent that has tested positive in animal experiments in more than one species, sex, strain, site or exposure route, with or without evidence of carcinogenicity in humans;

- "A positive tumor study that raises additional biological concerns beyond that of a statistically significant result, for example, a high degree of malignancy or an early age at onset;

- "A rare animal tumor response in a single experiment that is assumed to be relevant to humans; or

- "A positive tumor study that is strengthened by other lines of evidence."

According to the Guidelines, the descriptor "suggestive evidence of carcinogenicity":

"is appropriate when the weight of evidence is suggestive of carcinogenicity; a concern for potential carcinogenic effects in humans is raised, but the data are judged not sufficient for a stronger conclusion. This descriptor covers a spectrum of evidence associated with varying levels of concern for carcinogenicity, ranging from a positive cancer result in the only study on an agent to a single positive cancer result in an extensive database that includes negative studies in other species. Depending on the extent of the database, additional studies may or may not provide further insights. Some examples include:

- "A small, and possibly not statistically significant, increase in tumor incidence observed in a single animal or human study that does not reach the weight of evidence for the descriptor 'Likely to Be Carcinogenic to Humans;'

- "A small increase in a tumor with a high background rate in that sex and strain, when there is some but insufficient evidence that the observed tumors may be due to intrinsic factors that cause background tumors and not due to the agent being assessed;

- "Evidence of a positive response in a study whose power, design, or conduct limits the ability to draw a confident conclusion (but does not make the study fatally flawed), but where the carcinogenic potential is strengthened by other lines of evidence; or

- "A statistically significant increase at one dose only, but no significant response at the other doses and no overall trend."

b. Application of the Guidelines to TCE

In considering the data in the context of applying the "Carcinogenic to Humans" descriptor, one first considers the weight of the epidemiological evidence. We judge the epidemiologic evidence to be neither "convincing" nor "strong," two key terms in the Guidelines. This judgment is based on four recent reviews and meta-analyses of occupational TCE exposures and cancer as well as other reviews of this literature.[22] The recent review and meta-analysis by Kelsh *et al.* focuses on occupational TCE exposure and kidney cancer, and includes the Charbotel *et al.* study that is emphasized in the IRIS and Work Plan Assessments.[23] Both the EPA meta-analysis and the Kelsh *et al.* meta-analysis of the TCE kidney cancer epidemiologic literature produced similar summary results. However in Kelsh *et al.* the limitations of this body of research, namely exposure assessment limitations, potential unmeasured confounding, potential selection biases, and inconsistent findings across groups of studies, did not allow for a conclusion that there is sufficient evidence of a causal association, despite a modest overall association.

There are reasonably well-designed and well-conducted epidemiologic studies that report no association between TCE and cancer, some reasonably well-designed and conducted studies that did report associations between TCE and cancer, and finally some relatively poorly designed studies reporting both positive and negative findings. Overall, the summary relative risks or odds ratios in the meta-analysis studies (EPA or published meta-analyses) generally ranged between 1.2 and 1.4. The draft assessment refers to these associations as "small," a term not

[22] Alexander, D, *et al.*, A meta-analysis of occupational trichloroethylene exposure and multiple myeloma or leukaemia, Occup Med (Lond) 56:485–493 (2006); Alexander, D, *et al.*, A meta-analysis of occupational trichloroethylene exposure and liver cancer, Int Arch Occup Environ Health 81(2):127–43 (2007); Mandel, J, *et al.*, Occupational trichloroethylene exposure and non-Hodgkin's lymphoma: a meta-analysis and review, Occup Environ Med 63:597–607 (2006); Kelsh, M, *et al.*, Occupational trichloroethylene exposure and kidney cancer: a meta-analysis, Epidemiology 21(1): 95-102 (January 2010).

[23] Charbotel, B, *et al.*, Case-control study on renal cell cancer and occupational exposure to trichloroethylene, Part II: Epidemiological aspects, Ann Occup Hyg 50(8):777–787 (2006).

typically consistent with "convincing" and "strong." Weak or small associations may be more likely to be influenced by or be the result of confounding or bias. Smoking and body mass index are well-established risk factors for kidney cancer, and smoking and alcohol are risk factors for liver cancer, yet the potential impact of these factors on the meta-analysis associations was not fully considered. There were suggestions that these factors may have impacted findings (*e.g.*, in the large Danish cohort study of TCE exposed workers, the researchers noted that smoking was more prevalent among the TCE exposed populations, however little empirical data were provided). In addition, co-linearity of occupational exposures (*i.e.*, TCE exposure correlated with chemical and/or other exposures) may make it difficult to isolate potential effects of TCE from those of other exposures within a given study, and hinder interpretation across studies. For example, although Charbotel *et al.* reported potential exposure response trends, while controlling for many confounders of concern (which strengthens the weight of evidence), they also reported attenuated associations for cumulative TCE exposure after adjustment for exposure to cutting fluids and other petroleum oils (weakening the weight of the evidence). This study is also limited due to other potential study design considerations such as selection bias, self reporting of work histories, and residual confounding.

When examining the data for TCE and non-Hodgkin lymphoma, kidney cancer, and liver cancer, associations were inconsistent across occupational groups (summary results differed between aerospace/aircraft worker cohorts compared with workers from other industries), study design, location of the study, quality of exposure assessment (*e.g.*, evaluating studies that relied upon biomonitoring to estimate exposure *vs.* semi-quantitative estimates *vs.* self-report, etc.), and by incidence *vs.* mortality endpoints. Although EPA examined high dose categories, it did not evaluate any potential dose-response relationships across the epidemiologic studies (except for Charbotel *et al.*). Reviews of the epidemiologic data reported in various studies for different exposure levels (*e.g.*, cumulative exposure and duration of exposure metrics) did not find consistent dose-response associations between TCE and the three cancer sites under review.[24] An established dose-response trend is one of the more important factors when making assessments of causation in epidemiologic literature. Thus, based on an overall weight of evidence analysis of the epidemiologic research, these data do not support the conclusion that there is "strong" or "convincing" evidence of a causal association between human exposure and cancer.

EPA's Guidelines also state that a chemical may be described as "Carcinogenic to Humans" with a lesser weight of epidemiologic evidence that is strengthened by other lines of evidence, all of which must be met. One of these lines of evidence is "extensive evidence of carcinogenicity in animals." Therefore, we must briefly evaluate the animal data.

The criteria that have to be met for animal data to support a "carcinogenic to humans" classification are stated in a sequential manner with an emphasized requirement that all criteria have to be met. Since the Guidelines consider this to be an "exceptional" route to a "carcinogenic to humans" classification, we would expect rigor to have been applied in assessing animal data against the criteria. This simply was not done.

Of the four primary tissues that EPA evaluated for carcinogenicity, only one or perhaps two rise to the level of biological significance. Discussion of the remaining tumor types appears to presuppose

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[24] Mandel, J, *et al.*, Occupational trichloroethylene exposure and non-Hodgkin's lymphoma: a meta-analysis and review, Occup Environ Med 63:597–607 (2006); Alexander, D, *et al.*, A meta-analysis of occupational trichloroethylene exposure and liver cancer, Int Arch Occup Environ Health 81(2):127–43 (2007); Kelsh, M, *et al.*, Occupational trichloroethylene exposure and kidney cancer: a meta-analysis, Epidemiology 21(1): 95-102 (January 2010).

that TCE is carcinogenic. The resulting discussion appears then to overly discount negative data, of which there are many, and to highlight marginal findings. The text does not appear to be a dispassionate rendering of the available data. Specifically, EPA's conclusion that kidney cancer is evident in rats rests on *one* statistically significant finding in over 70 dose/tumor endpoint comparisons and references to exceedances of historical control values.[25] Using a 0.05 p-value for statistical significance, a frequency of 1 or even several statistically or biologically significant events is expected in such a large number of dosed/tumor groups. EPA's overall conclusion based on these flawed studies cannot be that TCE is a known kidney tumorigen. The best that can be said is that the data are inconsistent. Certainly they do not meet the criterion of "extensive evidence of carcinogenicity in animals." Several marginal findings do not constitute "extensive evidence."

For all these reasons, EPA's classification of TCE as "Carcinogenic to Humans" is not supported by the evidence and cannot be justified under the 2005 Guidelines.[26]

c. EPA's Position that there is 'Convincing Evidence' that TCE Is Carcinogenic to Humans is Inconsistent with National Academy of Sciences Conclusion of only 'Limited or Suggestive Evidence'

The IRIS Assessment states that "TCE is characterized as 'carcinogenic to humans' by all routes of exposure. This conclusion is based on convincing evidence of a causal association between TCE exposure in humans and kidney cancer."

Box 2 of the Academy's Camp Lejeune report, attached as Appendix 3, categorizes every cancer outcome reviewed in relation to exposure to TCE, the dry cleaning solvent perchloroethylene, or a mixture of the two. The categories are taken directly from a respected Institute of Medicine (IOM) report.[27] These categories are "sufficient evidence of a causal relationship," "sufficient evidence of an association," "limited or suggestive evidence of an association," "inadequate evidence to determine an association," and "limited or suggestive evidence of no association," all as defined in Box 1, also attached.

Looking at Box 2, evidence considered by EPA to be "convincing evidence of a causal association between TCE exposure in humans and kidney cancer" would seem to be considered "sufficient evidence of a causal relationship." Yet the Academy found no outcomes in that category. It would at least be "sufficient evidence of an association." Again, the Academy found no outcomes in that category. Only in the third category, "limited or suggestive evidence of an association," does one find kidney or any other cancer outcome associated with TCE.

"Limited evidence of an association" is far from "convincing evidence of causation." One would expect at the least a detailed explanation of EPA's very different conclusion. Although the 2009 Camp Lejeune study was already published, and indeed is cited in the references, there is no mention of it in the text of the IRIS Assessment, even though the previous draft had just been the subject of a multi-year review by the Academy.

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[25] And that bioassay is from a laboratory whose studies EPA has reviewed and declined to rely upon in other assessments.

[26] Further commentary to this effect, provided by a distinguished group of consultants in connection with the TCE IRIS Assessment but not addressed by EPA, is attached as Appendix 2.

[27] Institute of Medicine, Gulf War and Health, Vol. 2, Insecticides and Solvents (National Academies Press) (2003).

The Camp Lejeune committee began with a comprehensive review of the epidemiology studies of the two solvents by the IOM for its Gulf War Report. They then identified new studies published from 2003 to 2008 and considered whether these changed the conclusions in the IOM report. In the case of TCE and kidney cancer, this was the case. The Camp Lejeune committee considered six new cohort studies and two case-control studies (including Charbotel *et al.*). They concluded that several of these studies reported an increased risk of kidney cancer, but observed that the results were often based on a relatively small number of exposed persons and varied quality of exposure data and methodology. Given these data, the committee raised the classification for TCE to match the IOM conclusion of "limited" evidence for perchloroethylene.

EPA, on the other hand, offered the summary conclusion of convincing human evidence, based on the "consistency" of increased kidney cancer across the different studies. The authors of these studies, however, do not agree with EPA's characterization of them. For example, the authors of Charbotel *et al.*, the study EPA finds most compelling, state that the "study suggests an association between exposures to high levels of TCE and increased risk of [renal cell carcinoma]. Further epidemiological studies are necessary to analyze the effect of lower levels of exposure."

Given the flaws in the IRIS Assessment, and the very different conclusion reached by the Academy in its Camp Lejeune report on the same body of data, the Work Plan Assessment should not rely on the IRIS Assessment's classification of TCE as "Carcinogenic to Humans."

2. EPA Should Reassess Available Cancer Epidemiology Data, Given Publication of More Recent and Larger Studies on Worker Populations

The observation of an elevated but weak kidney cancer association reported by Charbotel *et al.* (2006)[28] contrasts with other occupational studies which did not find an elevation in kidney cancer in industries using TCE as a metal degreaser, *e.g.*, aircraft manufacturing, metal cleaning, etc., where exposures may be higher than for screw cutters. Lipworth and coworkers (2011) found no evidence of increased kidney cancer in a large worker cohort with multiple decades of TCE exposure and extended cancer follow-up evaluations.[29] The aircraft manufacturing study involved a total cohort of 77,943 workers, of which 5,443 were identified as exposed to TCE. The study involved evaluations from 1960 through 2008, at which time 34,248 workers had died. Approximately 30% of the workers were hired before 1960 (60% born before 1940), 52% terminated employment by 1980, and approximately a third of the workers were employed for more than 20 years. The standardized incidence ratio (SIR) for kidney cancer in the TCE-exposed workers was reported as 0.66 (CI 95%: 0.38-1.07). This value for the SIR indicates that these workers were potentially less likely to get kidney cancer than the normal population (or at least had the same rate as the normal population – SIR of 1).

More recently, two large Nordic country epidemiological studies, both of which had extensive follow-up of the cohorts, have likewise failed to find an association between TCE and kidney cancer. An SIR of 1.01 (0.70-1.42) was found by Hansen *et al.* (2013) for kidney cancer based on 32 cases out of a total of 997 cancer cases in a cohort of 5,553 workers in Finland, Sweden, and Denmark, indicating that

[28] Charbotel, B, *et al.*, Case-control study on renal cell cancer and occupational exposure to trichloroethylene, Part II: Epidemiological aspects, Ann Occup Hyg 50(8):777–787 (2006).

[29] Lipworth L, Sonderman JS, Mumma MT, *et al.*, Cancer mortality among aircraft manufacturing workers: an extended follow-up, J Occup Environ Med 53(9): 992-1007 (2011).

rates were the same as the normal population.[30] TCE exposures in this cohort were directly confirmed from urinary biomonitoring of the TCE metabolite trichloroacetic acid (TCA). However, overall TCE exposures were likely low in this cohort in that most urinary TCA measurements were less than 50 mg/L, corresponding to approximately 20 ppm TCE exposure. Thus, consistent with the conclusions of Bruning *et al.* (2003),[31] this study indicates TCE is unlikely to be a low-dose kidney carcinogen.

Similarly, no evidence of kidney cancer was found by Vlaanderen *et al.* (2013) in a recent follow-up examination of the Nordic Occupational Cancer cohort (Finland, Iceland, Norway, Sweden) in which statistically non-significant risk ratios (RR) of 1.01 (0.95-1.07), 1.02 (0.97-1.08), and 1.00 (0.95-1.07) were reported for a total of 4,145 renal cancer cases approximately equally distributed across three respective TCE exposure groups (tertiles) assigned from a job exposure matrix analysis.[32] Finally, although a meta-analysis of 23 studies meeting criteria for study inclusion found a slightly increased simple summary association of TCE and kidney cancer, RR 1.42 (1.17-1.77), more detailed analyses of subgroups suggested no association, or possibly a moderate elevation in kidney cancer risk, and no evidence of increasing risk with increasing exposure.[33]

These more recent studies were not reviewed in the 2011 TCE IRIS Assessment or the 2014 TCE Work Plan Assessment that form the basis for the proposed regulation. Any regulatory action under TSCA § 6, however, is required to be based on the "best available science" supported by "substantial evidence in the record." This provides compelling support for our position that the instant proposal should be withdrawn and the uses under consideration be examined following the TCE assessment EPA will be conducting in the near future under TSCA § 6(b)(4)(A).

3. EPA's Reliance on Charbotel *et al.* (2006) Results in an Overly Conservative Estimate of Risk

In its 2014 Work Plan Assessment of potential cancer risk, EPA focused solely on inhalation exposures and relied on an inhalation unit risk (IUR) value developed in the 2011 IRIS Assessment. The IUR was based primarily on epidemiology data from the case-control study on renal cell carcinoma (RCC) by Charbotel *et al.* (2006), discussed above. Although other epidemiological studies were used to derive an adjusted IUR estimate for the combined risk of developing RCC, NHL, or liver cancer, EPA concedes a lower level of confidence in both the NHL and liver cancer databases. While the Charbotel *et al.* study suggests a relationship between cumulative TCE exposure and RCC incidence, the reliability of the exposure estimates is a major concern.

The National Academy of Sciences Committee that reviewed the draft IRIS assessment released in 2001 recommended that:

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[30] Hansen J, Sallmén M, Seldén AI, *et al.*, Risk of cancer among workers exposed to trichloroethylene: analysis of three Nordic cohort studies, J Natl Cancer Inst 105(12): 869-877 (2013).

[31] Brüning T, Pesch B, Wiesenhütter B, *et al.*, Renal cell cancer risk and occupational exposure to trichloroethylene: Results of a consecutive case-control study in Arnsberg, Germany, Am J Ind Med. 43(3): 274-285 (2003).

[32] Vlaanderen J, Straif K, Pukkala E, *et al.*, Occupational exposure to trichloroethylene and perchloroethylene and the risk of lymphoma, liver, and kidney cancer in four Nordic countries, Occup Environ Med 70(6): 393-401 (2013).

[33] Kelsh MA, Alexander DD, Mink PJ, Mandel JH, Occupational trichloroethylene exposure and kidney cancer: a meta-analysis, Epidemiology 21(1): 95-102 (2010).

> "[t]here appear to be insufficient epidemiologic data to support quantitative dose-response modeling for trichloroethylene and cancer. The committee recommends that toxicologic data be used to fit the primary dose-response model(s) and that the available epidemiologic data be used only for validation. The committee does not believe that the available information is sufficient to determine the best dose-response model for trichloroethylene."[34]

EPA should follow the recommendation of the National Academy of Sciences, which referenced the Charbotel *et al.* (2005) final study report in its review of TCE.[35] The authors' own conclusions that the study only "suggests that there is a weak association between exposures to TRI [TCE] and increased risk of RCC" argues against the existence of the robust relationship which should be required for a dose-response assessment used as the basis for regulation.[36]

The exposure assessment for the Charbotel study was based on questionnaires and expert judgment, not direct measures of exposure.[37] Worker exposure data from deceased individuals were included in the study. In contrast to living workers, who were able to respond to the questionnaires themselves, exposure information from deceased workers (22.1% of cases and 2.2% of controls) was provided by surviving family members. The authors acknowledge that "this may have led to a misclassification for exposure to TCE due to the lower levels in the quality of information collected."

Analysis of the data revealed evidence of confounding from cutting fluid exposure. Unfortunately, TCE and cutting oil were co-exposures that could not be disaggregated and the majority of

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[34] National Research Council, Assessing the human health risks of trichloroethylene: key scientific issues, National Academies Press, Washington, DC (2006); http://www.nap.edu/openbook.php?record_id=11707&page=R1.

[35] Charbotel B, Fevotte J, Hours M, *et al.*, Case-control study on renal cell cancer and occupational trichloroethylene exposure, in the Arve Valley (France), Lyon, France: Institut Universitaire de Médecine du Travail, UMRESTTE, Université Claude Bernard (2005); http://hal.archives-ouvertes.fr/docs/00/54/59/80/PDF/charbotel_octobre_05.pdf

[36] This concern was recognized by the European Chemicals Agency (ECHA) in its 2013 Chemical Safety Report on TCE: "[T]here are several concerns with this study that should be taken into consideration when assessing its use in risk assessment and hazard characterization. For example, potential selection bias, the quality of the exposure assessment, and the potential confounding due to other exposures in the work place. With respect to the potential for selection bias, no cancer registry was available for this region to identify all relevant renal cell cancer cases from the target population. Case ascertainment relied on records of local urologists and regional medical centers; therefore, selection bias may be a concern. Given the concerns of the medical community in this region regarding renal cell cancer (RCC) among screw cutting industry workers, it is likely that any cases of renal cell cancer among these workers would likely be diagnosed more accurately and earlier. It is also much more unlikely that an RCC case among these workers would be missed compared to the chance of missing an RCC case among other workers not exposed to TCE. This preference in identifying cases among screw-cutting industry workers would bias findings in an upward direction. Concerning the potential for other exposures that could have contributed to the association, screw-cutting industry workers used a variety of oils and other solvents. Charbotel *et al.* reported lower risks for TCE exposure and renal cell cancer once data were adjusted for cutting oils. In fact, they noted, 'Indeed many patients had been exposed to TCE in screw-cutting workshops, where cutting fluids are widely used, making it difficult to distinguish between cutting oil and TCE effects.' This uncertainty questions the reliability of using data from Charbotel *et al.* since one cannot be certain that the observed correlation between kidney cancer and exposure is due to trichloroethylene."

[37] Fevotte J, Charbotel B, Muller-Beauté P, *et al.*, Case-control study on renal cell cancer and occupational exposure to trichloroethylene, Part I: Exposure assessment, Ann Occup Hyg 50: 765-775 (2006); http://dx.doi.org/10.1093/annhyg/mel040.

the TCE exposed population, the screw cutters, could be expected to experience similar patterns of exposure for both TCE and cutting fluids (probably in aerosol form). Thus the apparent dose-response relationship for TCE could be wholly or in part the result of exposure to cutting fluids.

In their 2006 publication of the study results, the authors assigned cumulative exposures into tertiles (i.e., low, medium and high), yet the dose-response evaluation conducted as part of the IRIS Assessment relied on mean cumulative exposure levels provided at a later date.[38] Although the IRIS Assessment references the email submission of the data to EPA, it provides no detail on the technical basis for the table, raising serious transparency issues.

In an apparent acknowledgement of the uncertainty of the exposure information, Charbotel *et al.* (2006) included an evaluation of "the impact of including deceased patients (proxy interviews) and elderly patients (>80 years of age)" on the relationship between exposure to TCE and RCC. Interestingly, it was stated that "only job periods with a high level of confidence with respect to TCE exposure were considered" in the study, an apparent reference to the use of two different occupational questionnaires, one "devoted to the screw-cutting industry and a general one for other jobs." As the Adjusted Odds Ratio (OR) for the high cumulative dose group was actually higher in the censored subgroup than in the uncensored group [3.34 (1.27-8.74) vs 2.16 (1.02-4.60)], the authors cavalierly suggested that "misclassification bias may have led to an underestimation of the risk."

What the authors and EPA appear to have overlooked is that, in addressing the misclassification bias, Charbotel may also have altered the cumulative dose-response relationship. For example, in the censored subgroup there were now only 16 exposed cases (1 in the Low Group, 4 in the Medium Group and 11 in the High Group) with Adjusted ORs of 0.85, 1.03 and 3.34, respectively. If the dose-response relationship in this higher-confidence subgroup has changed, use of the lower-confidence group to calculate the IUR would have to be rigorously justified by EPA before it could be considered sufficiently robust to drive the types of decisions based on unit risk that are found in the proposed rule.

4. Use of TCE Glutathione Conjugate Derived Metabolites Dichlorovinylglutathione (DCVG) and Dichlorovinylcysteine (DCVC) in TCE Renal Toxicity and Cancer Risk Assessment Should Be Reconsidered Given Improved Understanding of the Differential Quantitative Formation of these Metabolites in Animals Relative to the TCE Oxidative Metabolites Trichloroethanol (TCOH), Trichloroacetic Acid (TCA) and Dichloroacetic Acid (DCA)

The TCE IRIS Assessment relies in part on the conclusion that DCVG and DCVC, which are weakly active renal toxicants and genotoxicants, are formed in toxicologically significant concentrations following human exposures to TCE. Importantly, the basis for this conclusion rests on studies in which a relatively high blood DCVG concentration (100 nM) was observed in volunteers exposed for 4 hours to 50 or 100 ppm TCE.[39] However, Lash *et al.* (1999) relied on a colorimetric chromatographic method analysis of TCE glutathione conjugate-derived metabolites which had substantial potential for detection of non-TCE-specific endogenous substances. Subsequent radiochemical and HPLC-MS/MS based analyses that specifically quantitated both DCVG and DCVC have found that the activity of the

[38] Charbotel B (2008) [Email from Barbara Charbotel, University of Lyon, to Cheryl Scott, EPA].

[39] Lash LH, Putt DA, Identification of S-(1,2-dichlorovinyl)glutathione in the blood of human volunteers exposed to trichloroethylene, J Toxicol Env Hlth Part A, 56: 1-21 (1999).

glutathione conjugate pathway is substantially lower than that of the oxidative pathway resulting in TCA and DCA formation in both animals and humans.[40]

Since the publication of the TCE IRIS Assessment in 2011, additional studies have evaluated the kidney concentrations of TCE oxidative and glutathione conjugate-derived metabolites in a variety of mouse strains administered 5 daily oral 600 mg/kg doses of TCE.[41] Metabolites were quantitated 2 hr after the last daily dose in that toxicokinetic evaluations had shown the approximate maximum plasma concentrations of TCA, DCA, DCVG and DCVC were observed 2 hr following oral TCE treatment.[42] Using a structure-specific HPLC-ESI-MS/MS method, Yoo *et al.* (2015) demonstrated that DCVG and DCVC were only a very small fraction of total oxidative metabolites quantitated in kidney. TCOH kidney concentrations were 2-4-fold greater than TCA, and TCA concentrations were 100-1000 greater than DCA. Importantly, DCA concentrations were 100-1000-fold greater than DCVG and DCVC, resulting in the conclusion that TCE oxidative metabolism was up to 5 orders of magnitude greater than glutathione conjugate-derived metabolism. These findings were consistent with the earlier report from Kim *et al.* (2009) in which the plasma toxicokinetics TCA, DCA, DCVG and DCVC following a single 2140 mg/kg oral TCE dose found that the cumulative AUC of oxidative metabolites was 40,000-fold higher than the combined AUC of DCVG and DCVC; note that this study did not quantify TCOH, which would have further increased the disparity of glutathione conjugate-derived relative to oxidative-derived metabolites. These data demonstrate a dramatically lower function glutathione-conjugate metabolism relative to oxidative metabolism in mice, despite the observation by Dekant (2010) that mice generate DCVC at slightly higher rates than rats and greater than 10-fold higher than humans.

The results of studies using structure-specific analytical methods for quantitation of DCVG and DCVC directly challenge the hypothesis that glutathione conjugate-derived metabolites plausibly account for the genotoxicity, renal cytotoxicity, and ultimate carcinogenicity in rodents.[43] DCVC was only marginally cytotoxic (LDH release), if at all, when incubated at 0.2M (200,000 nM) with isolated renal cortical cells of male and female rats. This *in vitro* concentration is substantially higher than the approximate maximum kidney concentrations of 10-75 nM DCVC resulting from treatment of various strains of mice with a high oral TCE dose of 600 mg/kg/day for 5 days observed by Yoo *et al.* (2015). In addition, a likely NOAEL of 1 mg/kg/day was reported for kidney toxicity (no change in serum BUN, weak tubule dilation and no necrosis) in mice administered DCVC orally or intraperitoneally at 1, 10 or 30 mg/kg/day, 1 day per week, for 13 weeks.[44] If, based on Yoo *et al.* (2015), it is assumed that the ratio of formation of oxidative metabolites to glutathione conjugate-derived metabolites is 10,000:1, an implausibly high (occupational or general population) dose of 6044 mg/kg TCE would be required to

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[40] Dekant,W (2010), attached as Appendix 4.

[41] Yoo HS, Bradford BU, Kosyk O, Uehara T, Shymonyak S, Collins LB, Bodnar WM, Ball LM, Gold A, Rusyn I, Comparative analysis of the relationship between trichloroethylene metabolism and tissue-specific toxicity among inbred mouse strains: kidney effects, J Toxicol Env Hlth Pt A, 78: 32-49.b (2015).

[42] Kim, S, Kim, D, Poilack, GM, Collins, LB, and Rusyn, I, Pharmacokinetic analysis of trichloroethylene metabolism in male B6C3F1 mice: Formation and disposition of trichloroacetic acid, dichloroacetic acid, S-(1,2-dichlorovinyl)glutathione and S-(1,2-dichlorovinyl)-L-cysteine, Toxicol Appl Pharmacol 238: 90-99 (2009).

[43] Lash LH, Qian W, Putt DA, Hueni SE, Elfarra AA, Krause RJ, Parker JC, Renal and hepatic toxicity of trichloroethylene and its glutathione-derived metabolites in rats and mice: Sex-, species-, and tissue-dependent differences, J Pharmacol Exp Ther 297: 155-164 (2001).

[44] Shirai N, Ohtsuji M, Hagiwara K, Tomisara H, Ohtsuje N, Hirose S, Hagiwara H, Nephrotoxic effect of subchronic exposures to S-(1,2-dichlorovinyl)-L-cysteine in mice, J Toxicol Sci 37: 871-878.h (2012).

deliver a NOAEL dose of 1 mg/kg/day DCVC (1 mmol/kg/day TCE results in 0.0001 mmol/kg/day DCVC; 1 mg/kg/day DCVC = 0.0046 mmol/kg/day). These dose-toxicity calculations suggest that it appears toxicologically implausible that real-world exposures to TCE are capable of producing doses of DCVC sufficient to cause renal toxicity and carcinogenicity in mice.

D. Peer Review Ignored

The draft Work Plan Assessment was the subject of peer review by a panel selected by EPA in 2013. The peer review report highlights that it was a screening level assessment that inappropriately relied on an unreproducible study, and recommended that the assessment be abandoned.[45] One reviewer devoted six pages to a very detailed critique of Johnson *et al.* (2003) and EPA's reliance on such a deficient study.[46] Nevertheless, EPA ignored the peer review. Remarkably, even though the trade press article on the peer review was entitled *EPA Peer Reviewers Say Trichloroethylene Analysis Not Ready for Regulatory Use*, the EPA Assistant Administrator for Chemical Safety and Pollution Prevention wrote to the EPA Inspector General that "[i]t is notable that *the external peer reviews of all the Work Plan assessments we have completed thus far supported our overall assessment methodologies and conclusions*."[47] A more detailed description of the peer reviewers' comments is attached as Appendix 3.

Ppeer review is identified as a key step in EPA's proposed procedures for chemical risk evaluation under TSCA as amended. EPA states that "[i]n addition to any targeted peer review of specific aspects of the analysis, the entire risk assessment will also undergo peer review, as it is important for peer reviewers to consider how the various underlying analyses fit together to produce an integrated risk characterization which will form the basis of unreasonable risk determination."[48] As the draft Work Plan Assessment for TCE did not address the spot cleaning scenario at all, the assessment of risks under that scenario has *never* been subjected to peer review. Thus an applicable requirement of TSCA §§ 6 and 26(l)(4) for reliance on the Work Plan Assessment has not been met.

E. Screening Level Assessment

As noted above and in Appendix 5, the peer review report highlights that the Work Plan Assessment was a screening level assessment. Specifically, the Chairperson of EPA's peer review panel wrote:

> "The draft document fails to articulate satisfactorily that the analysis described within should be characterized as a screening level assessment. . . . I believe that the Agency acted prematurely in issuing this (screening level) assessment for public comment. . . . After listening carefully to the comments and contributions from the other members of

[45] https://www.epa.gov/sites/production/files/2015-09/documents/tce_consolidated_peer_review_comments_september_5_2013.pdf.

[46] *Id.*

[47] Response to Office of Inspector General Draft Report No. OPE-FY14-0012 "EPA's Risk Assessment Division Has Not Fully Adhered to Its Quality Management Plan," (July 30, 2014), Appendix A, p.10 (available at https://www.epa.gov/sites/production/files/2015-09/documents/20140910-14-p-0350.pdf) (emphasis added). *Compare* BNA Daily Environment Report, *EPA Peer Reviewers Say Trichloroethylene Analysis Not Ready for Regulatory Use* (July 18, 2013).

[48] 82 Fed. Reg. at 7572.

the Panel, I have concluded that there would little benefit in revising this draft screening assessment."

With regard to aerosol degreasing, EPA identified only two aerosol degreasing products containing TCE in the marketplace and found no emissions or monitoring data for either product – thus these are hypothetical exposures. Further, EPA used E-FAST2/CEM modeling to develop "high-end acute inhalation exposure estimates" based solely on professional judgment, providing confirmation that this is a screening level assessment. The highest uncertainties were associated with mass of product used per event, duration of event, and number of events per year, as the values selected were hypothetical, thus leading to further lack of confidence in the assessment.

For spot cleaning workers the problems with the exposure assessment are even more obvious. A major limitation of the exposure assessment used to evaluate potential risk arising from spot cleaning operations was the unavailability of relevant exposure monitoring data. Section 2.4.2.5 of the Work Plan Assessment, however, references a study specific to spot cleaning and states that "site-specific parameters from this study were incorporated into the NF/FF model to obtain site-specific model estimates of worker exposure."[49]

Examination of the NIOSH (1997) study reveals that the air monitoring was actually conducted in response to an OSHA complaint from workers and the report states that "[c]onditions at this shop were probably worst case." Use of monitoring data from a worst case, potential enforcement situation adds additional strength to the concern that the Work Plan Assessment is actually a screening level assessment which does not reflect normal operating conditions and exposures.

It is clear that a risk evaluation that supports a TSCA § 6 rule must be more robust than the screening level Work Plan Assessment that EPA carried out for TCE, which does not comply with Office of Management and Budget ("OMB") guidelines implementing the Information Quality Act.[50] First, EPA must conduct a "highly influential scientific assessment" to support TSCA § 6 rulemaking. OMB defines a scientific assessment as "highly influential" if dissemination of the assessment could have a potential impact of more than $500 million in any one year on either the public or private sector, or if the dissemination is novel, controversial, precedent-setting, or has significant interagency interest.

The TCE assessment employed worst-case or default assumptions that led to overestimation of potential risks. Such assessments may be appropriate to support a decision that no further action or evaluation is necessary, because there is confidence that the potential risks are not a concern. However, they are inappropriate to support regulations intended to reduce risk because screening level assessments do not accurately estimate risk or quantify exposures. Second, OMB's guidelines also require agencies to subject highly influential scientific assessments to more rigorous peer review. For TCE, EPA selected a contractor to manage the peer review process, even though experts consider contractor-managed peer review to be the least rigorous level of peer review.

F. Summary of Concerns

[49] National Institute for Occupational Safety and Health (NIOSH), Control of Health and Safety Hazards in Commercial Dry Cleaning, Publication Number 97-150, Centers for Disease Control and Prevention, Atlanta, GA (1997); http://www.cdc.gov/niosh/docs/97-150/#controls

[50] OMB, Final Information Quality Bulletin for Peer Review (Dec. 16, 2004) (available at https://www.whitehouse.gov/sites/default/files/omb/assets/omb/memoranda/fy2005/m05-03.pdf).

In sum, the TCE Work Plan Assessment is inconsistent with the applicable requirements of revised § 6 in the following ways, among others:

- It expressly relies on hazard values derived directly from a single academic study to estimate non-cancer risk, even though several other studies, including two GLP-compliant studies conducted under EPA guidelines, have been unable to reproduce the effect;[51]

- The University of Arizona study upon which EPA relies has been heavily criticized in the published literature,[52] and other regulatory agencies have expressly declined to rely on the academic study citing data quality concerns;[53]

- The authors of the Arizona study have published repeated corrections that fail to address the data quality concerns;[54] and a majority of EPA's own staff scientists expressed "low" confidence in its results.[55]

- The Work Plan Assessment relies on qualitative and quantitative estimates of cancer risk that are not realistic or justified by any underlying science. Two recent large Nordic epidemiological studies, both of which had extensive follow-up of the cohorts, failed to find an association between TCE and kidney cancer, but these are not addressed in the Work Plan Assessment. Further, EPA's reliance upon a potency factor based on Charbotel *et al.* (2006) directly contravenes the advice EPA received from the National Academy of Sciences

- For aerosol degreasing EPA provided no emissions or monitoring data – thus these are hypothetical exposures. The spot cleaning exposure assessment relies solely on a 2007 California study, which EPA recognized may not be representative of US dry cleaning facilities. The draft TCE Assessment, entitled "Degreaser and Arts/Crafts Uses," did not address spot cleaning at all (except to say that none of those sold to consumers contained TCE), but the final Work Plan Assessment is entitled "Degreasing, *Spot Cleaning* and Arts & Crafts Uses" and includes commercial use of TCE as a spotting agent at dry cleaning facilities.

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[51] *Compare* Johnson *et al.* (2003) to Fisher, J, *et al.*, Trichloroethylene, trichloroacetic acid, and dichloroacetic acid: do they affect fetal rat heart development? Int. J. Toxicol. 20: 257-67 (2001) and Carney, E, *et al.*, Developmental toxicity studies in Crl:Cd (SD) rats following inhalation exposure to trichloroethylene and perchloroethylene, Birth Defects Research (Part B) 77: 405–412 (2006).

[52] *E.g.*, "Johnson and Dawson, with their collaborators, are alone in reporting that TCE is a 'specific' cardiac teratogen." Hardin, B, *et al.*, Trichloroethylene and cardiac malformations, Environ. Health Perspect. 112: A607-8 (2004); Watson, R., *et al.*, Trichloroethylene-contaminated drinking water and congenital heart defects: a critical analysis of the literature, Repro. Toxicol. 21: 117-47 (2006).

[53] *E.g.*, "The data from this study were not used to calculate a public-health protective concentration since a meaningful or interpretable dose-response relationship was not observed. These results are also not consistent with earlier developmental and reproductive toxicological studies done outside this lab in mice, rats, and rabbits." California EPA Public Health Goal for Trichloroethylene in Drinking Water (July 2009), at 21.

[54] Johnson, PD, *et al.*, Environ Health Perspect 122: A94 (2014): erratum to Johnson, PD, *et al.*, Environ Health Perspect 113:A18 (2005), which is an erratum to Johnson *et al.* (2003).

[55] TCE Developmental Cardiac Toxicity Assessment Update (available at http://www.regulations.gov/#!documentDetail;D=EPA-HQ-OPPT-2012-0723-0045).

- It is a screening level assessment which does not meet OMB guidelines implementing the Information Quality Act for a "highly influential scientific assessment" to support TSCA § 6 rulemaking.

- The report of the peer review of the TCE Assessment highlights the foregoing points in the clearest possible terms, but EPA ignored it.[56] In fact, the EPA Assistant Administrator characterized the peer review as supportive.

Following enactment of the Lautenberg Act, it should be clear that a risk evaluation that supports a TSCA § 6 rule must be more robust than the screening level Work Plan Assessment that EPA conducted for TCE. Peer review and public comments identified numerous scientific deficiencies with the draft assessment, including the inappropriate use of default assumptions; ignoring contrary evidence that affects the weight of the scientific evidence; reliance on inapposite exposure data; conclusions inconsistent with the evidence cited; and reliance on a study that is not reproducible. Important shortcomings in both the hazard and exposure assessments were noted. Whatever "best available science" may mean, it cannot include reliance on an unreproducible toxicity study, a cancer risk assessment that does not take into account relevant epidemiological and toxicological studies, or outdated and unrepresentative exposure information.[57] And certainly EPA can no longer afford to ignore the conclusions of the peer review it initiated, as TSCA § 26(h) requires it to consider "the extent of independent verification or peer review of the information."

II. Failure to Comply with SBREFA

The Regulatory Flexibility Act, as amended by the Small Business Regulatory Enforcement Fairness Act (SBREFA), provides:

> "(a) When any rule is promulgated which will have a significant economic impact on a substantial number of small entities, the head of the agency promulgating the rule or the official of the agency with statutory responsibility for the promulgation of the rule shall assure that small entities have been given an opportunity to participate in the rulemaking for the rule through the reasonable use of techniques such as—
>
> (1) the inclusion in an advance notice of proposed rulemaking, if issued, of a statement that the proposed rule may have a significant economic effect on a substantial number of small entities;
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> (2) the publication of general notice of proposed rulemaking in publications likely to be obtained by small entities;
>
> (3) the direct notification of interested small entities;

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[56] https://www.epa.gov/sites/production/files/2015-09/documents/tce_consolidated_peer_review_comments_september_5_2013.pdf.

[57] *See* 162 Cong. Rec. S3522 (June 7, 2016) ("For far too long Federal agencies have manipulated science to fit predetermined political outcomes, hiding information and underlying data, rather than using open and transparent science to justify fair and objective decision making. This Act seeks to change all of that and ensure that EPA uses the best available science, bases scientific decisions on the weight of the scientific evidence rather than one or two individual cherry-picked studies, and forces a much greater level of transparency that forces EPA to show their work to Congress and the American public.)"

(4) the conduct of open conferences or public hearings concerning the rule for small entities including soliciting and receiving comments over computer networks; and

(5) the adoption or modification of agency procedural rules to reduce the cost or complexity of participation in the rulemaking by small entities.

"(b) Prior to publication of an initial regulatory flexibility analysis which a covered agency is required to conduct by this chapter—

(1) a covered agency shall notify the Chief Counsel for Advocacy of the Small Business Administration and provide the Chief Counsel with information on the potential impacts of the proposed rule on small entities and the type of small entities that might be affected;

(2) not later than 15 days after the date of receipt of the materials described in paragraph (1), the Chief Counsel shall identify individuals representative of affected small entities for the purpose of obtaining advice and recommendations from those individuals about the potential impacts of the proposed rule;

(3) the agency shall convene a review panel for such rule consisting wholly of full time Federal employees of the office within the agency responsible for carrying out the proposed rule, the Office of Information and Regulatory Affairs within the Office of Management and Budget, and the Chief Counsel;

(4) the panel shall review any material the agency has prepared in connection with this chapter, including any draft proposed rule, collect advice and recommendations of each individual small entity representative identified by the agency after consultation with the Chief Counsel, on issues related to subsections 603(b), paragraphs (3), (4) and (5) and 603(c);

(5) not later than 60 days after the date a covered agency convenes a review panel pursuant to paragraph (3), the review panel shall report on the comments of the small entity representatives and its findings as to issues related to subsections 603(b), paragraphs (3), (4) and (5) and 603(c), provided that such report shall be made public as part of the rulemaking record; and

(6) where appropriate, the agency shall modify the proposed rule, the initial regulatory flexibility analysis or the decision on whether an initial regulatory flexibility analysis is required."[58]

No Small Business Advisory Review (also referred to as "SBREFA Panel") was held for the proposed rule, however. Instead, EPA determined and certified that the rule would "not, if promulgated, have a significant economic impact on a substantial number of small entities." Where such a certification is made, no initial or final regulatory analysis is required, and thus a SBREFA Panel need not be convened.[59]

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[58] 5 U.S.C. § 609(a), (b).

[59] 5 U.S.C. § 605(b): "Sections 603 and 604 of this title shall not apply to any proposed or final rule if the head of the agency certifies that the rule will not, if promulgated, have a significant economic impact on a substantial number of small entities. If the head of the agency makes a certification under the preceding sentence, the agency

HSIA submits that EPA could not lawfully have certified that the proposed rule banning the use of TCE in spot cleaning lacked SISNOSE. EPA has adopted guidance on making the SISNOSE determination:

> "The lower economic impact threshold is particularly important because it is used to screen out rules that generally will not have a significant economic impact and, therefore, can be presumed not to require an IRFA/FRFA (i.e., if all small entities subject to a rule face economic impacts less than the lower threshold, then the rule may be assigned to the Presumed No SISNOSE Category). For this reason the lower economic impact threshold should be set conservatively, at a level that precludes any reasonable possibility that a rule placed in the Presumed No SISNOSE Category might later be found to impose a "significant economic impact on a substantial number of small entities." The upper threshold defines a level of economic impact that would be unquestionably significant for a small entity. In analyzing previous rules, EPA has often defined the lower threshold as compliance costs of 1% of sales and the higher threshold as compliance costs of 3% of sales as shown in the example in Table 2."[60]

The guidance further states that where the number of small entities subject to the rule and experiencing given economic impact is 1,000 or more, regardless of the percentage these constitute of all the small entities subject to the rule that are experiencing given economic impact, the rule will be presumed ineligible for certification.[61]

Spot cleaning is conducted by dry cleaners, virtually all of which are small businesses. The National Cleaners Association (NCA) estimates that there are some 23,550 retail dry cleaning establishments in the United States, having average sales of $250,797 and average profits of $17,809. Industry suppliers report that 60-90% of retail dry cleaners routinely order TCE for use on the spotting board (14,130 – 21,195 small businesses).

During an EO 12866 meeting on October 3, 2016, NCA provided the foregoing and following information. TCE is one of the most used spotting agents. TCE's effectiveness as a spot remover helps cleaners minimize time spent in stain removal and therefore control labor and operational costs. In most small dry cleaning plants the stain removal technician is the highest paid employee. Depending on the operation, labor represents 25-42% (average 30%) of the dry cleaners' costs. Assuming that only twelve garments a day require five additional minutes of stain removal time, this will add one hour a day to the spotter's labor. Assuming the spotter earns just $35,000 per year, one extra hour per day in a 6-day week, with overtime involved, will result in an extra $7,875 in the spotter's gross wages. It will also result in increased utilities due to six additional hours per week of boiler time and plant operation. It will also result in wasted or slowed production in the pressing department as they wait longer for cleaned

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shall publish such certification in the Federal Register at the time of publication of general notice of proposed rulemaking for the rule or at the time of publication of the final rule, along with a statement providing the factual basis for such certification."

[60] Final Guidance for EPA Rulewriters: Regulatory Flexibility Act as amended by the Small Business Regulatory Enforcement Fairness Act, https://www.epa.gov/sites/production/files/2015-06/documents/guidance-regflexact.pdf, Table 2.

[61] *Id.*

garments, further increasing labor costs. Between labor and utilities, NCA estimated *an increased cost of between 4–5% of gross sales.*[62]

Even the lowest increased cost estimated by NCA (4% of gross sales), at the low end of the range of small dry cleaning entities (14,130), constitutes SISNOSE as defined in EPA's guidance. The economic analysis in the docket acknowledges a much larger universe of dry cleaning that use spot removers (48,602) but concludes, with no factual support, that all of these are expected to experience cost impacts that are less than one percent of their revenues.[63]

Remarkably, neither the preamble to the proposed rule nor the economic analysis contains a detailed "statement providing the factual basis for such certification [of no SISNOSE] required by law." Rather, the latter includes a remarkably abstruse discussion of "market failure" that could be inserted into any analysis to support regulation in the absence of data specific to an industry or small business sector.[64] It is respectfully submitted that this does not meet the requirements of the Regulatory Flexibility Act.

III. Failure to Comply with Notice Requirements of TSCA and Administrative Procedure Act

EPA's TCE Work Plan Assessment is legally deficient in a more fundamental way. The draft Assessment was entitled "Degreaser and Arts/Crafts Uses." It states that "EPA focused the assessment on uses of TCE as a degreaser (i.e., both in small commercial settings and by consumers or hobbyists) and on consumer use of TCE in products used by individuals in the arts and crafts field" (p. 14). Spot cleaning is mentioned only in fn. 8: "there were several spot cleaners for fabrics marketed to consumers, but none contained TCE; lists of ingredients were not available for a few of the spot cleaners." There was no reference at all to spot cleaning in the workplace. Yet, with no explanation, the final TCE Work Plan Assessment is entitled "Degreasing, *Spot Cleaning* and Arts & Crafts Uses" and includes "Commercial use of TCE as a spotting agent at dry cleaning facilities" (p. 26).

The failure to notify dry cleaners that EPA was assessing a key agent upon which they rely clearly violates TSCA § 6(b)(4)(H), which states: "The Administrator shall provide no less than 30 days

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[62] https://www.reginfo.gov/public/do/viewEO12866Meeting?viewRule=true&rin=2070-AK03&meetingId=2352&acronym=2070-EPA/OCSPP

[63] *See* Economic Analysis of Proposed TSCA Section 6 Action on Trichloroethylene in Dry Cleaning Spot Removers and Aerosol Degreasers, at ES-15. The difference in number of establishments is due to EPA's reliance on data from decades ago when dry cleaning was a much larger sector.

[64] It begins:

> "Market failure can justify government regulation; the major types of market failures include the following:
>
> - Negative externalities, common property resources, and public goods;
> - Market power;
> - Inadequate or asymmetric information.
>
> The occurrence of any of these conditions justifies further inquiry into the need for government regulation to reduce inefficiencies in the allocation of society's resources. This section describes why negative externalities and inadequate or asymmetric information are present in the market for dry cleaning spot removers and aerosol degreasing products."

Id. at 2-2.

public notice and an opportunity for comment on a draft risk evaluation prior to publishing a final risk evaluation." That this is an "applicable requirement[] of § 6" for purposes of TSCA § 26(l)(4), which sets forth the requirements for EPA to rely upon risk assessments completed prior to enactment of the Lautenberg Act, should be obvious. In addition, § 553 of the Administrative Procedure Act (APA) requires all federal agencies to provide public notice and an opportunity for comment on all proposed rules.[65] The APA definition of "rule" is broad and encompasses background data upon which the rule is based.

Because there was no notice that EPA was addressing spot cleaning, there was no participation by dry cleaner representatives and no peer review of the spot cleaning assessment. EPA based estimates of workers/bystanders on census data "not adjusted to exclude job categories that likely would not be present at dry cleaning facilities. Thus, EPA's estimate likely overestimates the size of the population exposed."[66] Moreover, EPA relied solely on a 2007 California study, which it recognized may not be representative of US dry cleaning facilities. As dry cleaners had no notice that EPA was assessing spot cleaning in the workplace, they did not have an opportunity to comment on the exposure estimates or the study. Thus, the minimal requirements of administrative procedure have not been met in this rulemaking.

An equally serious notice issue is presented by EPA's acknowledgement that it only evaluated the commercial use of TCE for spot cleaning at dry cleaning facilities in the final Work Plan Assessment in response to a peer reviewer comment. It is therefore obvious that the evaluation of this additional use in the final risk assessment was not itself actually peer reviewed. Similarly, the supplemental analyses conducted by EPA to identify risks for the commercial aerosol degreasing use scenario and for various parameters of exposure scenarios for TCE spot cleaner use in dry cleaning facilities were only done long after completion of the Work Plan Assessment and after passage of the Lautenberg Act. Further, these analyses have not been peer reviewed. As noted above, peer review of these analyses is required by the OMB Final Information Quality Bulletin for Peer Review and TSCA.

IV. EPA's Reliance on Alternatives is Unrealistic

TSCA § 6(c)(2) provides:

> "(C) CONSIDERATION OF ALTERNATIVES.—
>
> "Based on the information published under subparagraph (A), in deciding whether to prohibit or restrict in a manner that substantially prevents a specific condition of use of a chemical substance or mixture, and in setting an appropriate transition period for such action, the Administrator shall consider, to the extent practicable, whether technically and economically feasible alternatives that benefit health or the environment, compared to the use so proposed to be prohibited or restricted, will be reasonably available as a substitute when the proposed prohibition or other restriction takes effect."

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[65] 5 U.S.C. § 553(b), (c): "General notice of proposed rulemaking shall be published in the Federal Register, unless persons subject thereto are named and either personally served or otherwise have actual notice thereof in accordance with law. . . . After notice required by this section, the agency shall give interested persons an opportunity to participate in the rulemaking through submission of written data, views, or arguments with or without opportunity for oral presentation."

[66] TCE Work Plan Assessment, at 116.

The proposal suggests that n-propyl bromide (nPB), perchloroethylene, methylene chloride, and water-based compounds could be used as alternatives to TCE in spot cleaning. Many of these alternatives are ineffective, hence the continued market dominance of the TCE-based products. Moreover, there is serious question whether a number of these alternatives would realistically be available, given the designation of nPB, perchloroethylene, and methylene chloride as priorities for risk evaluation/regulation under TSCA § 6(b)(2)(A).[67]

Query how compounds such as nPB could be considered a "reasonably available" substitute for TCE, much less how EPA could consider making such a finding in light of the fact that substitution on nPB in foam fabrication following reduction of the workplace limit for methylene chloride is regarded as a textbook example of "regrettable substitution." Unlike TCE, which has a long history of safe use in the workplace, the serious health impairments suffered by workers in those facilities have been widely documented. Moreover, an nPB industry representative stated at EPA's February 14, 2017 meeting on scoping documents for the ten priority compounds that nPB is no longer used in dry cleaning at all.

V. Gap Filling Purpose of TSCA

As originally enacted and as updated by the Lautenberg Act, TSCA requires EPA to consult and coordinate with other federal agencies "for the purpose of achieving the maximum enforcement of this Act while imposing the least burdens of duplicative requirements on those subject to the Act and for other purposes."[68] Worker and consumer health and safety fall under the jurisdictions, respectively, of OSHA and the Consumer Product Safety Commission (CPSC). The use of TCE in spot cleaning and aerosol degreasing is already more than adequately regulated under the OSH Act and/or the Federal Hazardous Substances Act. This comprehensive regulatory framework provides adequate protections with respect to the same potential adverse impacts and potential exposure pathways targeted by the proposed rule. Taking steps that may lead to the removal of products from the marketplace because workers or consumers failed to comply with the existing legal requirements is not consistent with TSCA either as initially enacted or as revised.

The basis for EPA's broad assertion of jurisdiction over occupational and consumer uses is unclear. The Lautenberg Act eliminated the requirement in TSCA § 6(a) that EPA protect "against [unreasonable] risk using the least burdensome requirements," but did not materially change the existing framework that requires unreasonable risks to be addressed under statutory authority other than TSCA wherever possible. EPA's longstanding interpretation of this framework is as follows:

> "Under section 9(a)(1) of TSCA, the Administrator is required to submit a report to another Federal agency when two determinations are made. The first determination is that the Administrator has reasonable basis to conclude that a chemical substance or mixture presents or will present an unreasonable risk of injury to health or the environment. The second determination is that the unreasonable risk may be prevented or reduced to a sufficient extent by action taken by another Federal agency under a Federal law not administered by EPA. Section 9(a)(1) provides that where the Administrator makes these two determinations, EPA must provide an opportunity to the other Federal agency to assess the risk described in the report, to interpret its own statutory authorities, and to initiate an action under the Federal laws that it administers.
>
> "Accordingly, section 9(a)(1) requires a report requesting the other agency: (1) To determine if the risk may be prevented or reduced to a sufficient extent by action taken

[67] 81 Fed. Reg. 91927 (Dec. 19, 2016).

[68] TSCA § 9(d).

under its authority, and (2) if so, to issue an order declaring whether or not the activities described in the report present the risk described in the report.

"Under section 9(a)(2), EPA is prohibited from taking any action under section 6 or 7 with respect to the risk reported to another Federal agency pending a response to the report from the ether Federal agency. There would be no similar restriction on EPA for any risks associated with a chemical substance or mixture that is not within the section 9(a)(1) determinations and therefore not part of the report submitted by EPA to the other Federal agency."[69]

It was clear from the outset that TSCA is to be used only when other statutes fail to provide a remedy for unreasonable risks. When TSCA was enacted in 1976, Representative James Broyhill of North Carolina indicated that "it was the intent of the conferees that the Toxic Substance Act not be used, when another Act is sufficient to regulate a particular risk."[70] TSCA § 9(a) is substantively unchanged by the Lautenberg Act. The House Energy and Commerce Committee Report states: "H.R. 2576 reinforces TSCA's original purpose of filling gaps in Federal law that otherwise did not protect against the unreasonable risks presented by chemicals," and further clarifies that "while § 5 makes no amendment to TSCA § 9(a), the Committee believes that the Administrator should respect the experience of, and defer to other agencies that have relevant responsibility such as the Department of Labor in cases involving occupational safety."[71]

Colloquies on the floor of the House of Representatives make this intent clear with specific reference to TCE, most notably the following:

"Mr. SHIMKUS. Mr. Speaker, I yield 2 minutes to the gentlewoman from Tennessee (Mrs. *Blackburn*), the vice chair of the full committee.

Mrs. BLACKBURN. Mr. Speaker, I do rise in support of the amendments to H.R. 2576, and I congratulate Chairman *Shimkus* on the wonderful job he has done. Mr. Speaker, I yield to the gentleman from Illinois (Mr. *Shimkus*) for the purpose of a brief colloquy to clarify one important element of the legislation.

Mr. Chairman, it is my understanding that this bill reemphasizes Congress' intent to avoid duplicative regulation through the TSCA law. It does so by carrying over two important EPA constraints in section 9 of the existing law while adding a new, important provision that would be found as new section, 9(b)(2).

It is my understanding that, as a unified whole, this language, old and new, limits the EPA's ability to promulgate a rule under section 6 of TSCA to restrict or eliminate the use of a chemical when the Agency either already regulates that chemical through a different statute under its own control and that authority sufficiently protects against a risk of injury to human health or the environment, or a different agency already regulates that chemical in a manner that also sufficiently protects against the risk identified by EPA.

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[69] 4,4'-Methylenedianiline; Decision to Report to the Occupational Safety and Health Administration, 50 Fed. Reg. 27674 (July 5, 1985). EPA also has acted under § 9(a) to refer 1,3-butadiene and glycol ethers to OSHA, 50 Fed. Reg. 41393 (Oct. 10, 1985) and 51 Fed. Reg. 18488 (May 20, 1986), respectively, and to refer dioxins in bleached wood pulp and paper products to the Food and Drug Administration, 55 Fed. Reg. 53047 (Dec. 26, 1990).

[70] 122 Cong. Rec. H11344 (Sept. 28, 1976).

[71] H. Rep. No. 114-176 (114th Cong., 1st Sess.) at 28.

Would the chairman please confirm my understanding of section 9?

Mr. SHIMKUS. Will the gentlewoman yield?

Mrs. BLACKBURN. I yield to the gentleman from Illinois.

Mr. SHIMKUS. The gentlewoman is correct in her understanding.

Mrs. BLACKBURN. I thank the chairman. The changes you have worked hard to preserve in this negotiated bill are important. As the EPA's early-stage efforts to regulate methylene chloride and TCE under TSCA statute section 6 illustrate, they are also timely.

EPA simply has to account for why a new regulation for methylene chloride and TCE under TSCA is necessary since its own existing regulatory framework already appropriately addresses risk to human health. New section 9(b)(2) will force the Agency to do just that.

I thank the chairman for his good work."[72]

Indeed, TSCA § 9 was strengthened by the Frank R. Lautenberg Chemical Safety for the 21st Century Act, and it was clear from the outset that TSCA is to be used only when other statutes fail to provide a remedy for unreasonable risks. Representative James Broyhill of North Carolina indicated that "it was the intent of the conferees that the Toxic Substance Act not be used, when another act is sufficient to regulate a particular risk."[73] EPA applied this statutory directive in determining that the risk from 4,4' methylenedianiline (MDA) could be prevented or reduced to a significant extent under the Occupational Safety and Health Act, and referring the matter for action by OSHA.[74] And in an analysis of TSCA § 9, EPA's Acting General Counsel concluded that "Congress expected EPA – particularly where the Occupational Safety and Health Act was concerned – to err on the side of making referrals rather than withholding them."[75]

There is no evidence that EPA has submitted to OSHA "a report which describes such risk and includes in such description a specification of the activity or combination of activities which the Administrator has reason to believe so presents such risk and includes in such description a specification of the activity or combination of activities which the Administrator has reason to believe so presents such risk," as required by TSCA § 9(a)(1). The non-existent report obviously did not "include a detailed statement of the information on which it is based" and was not "published in the Federal Register," as required.

Had the required report been issued, it presumably would have identified how OSHA's authority over the workplace was insufficient to address the risks posed by spot cleaning and aerosol degreasing using TCE. A letter from the Assistant Secretary of Labor for Occupational Safety and Health (undated but apparently issued on April 4, 2016) identifies limits on OSHA's authority to regulate hazardous substances such as TCE, but it does not come close to meeting the requirements of TSCA for EPA action in this case. The April 2016 letter identifies no gap specific to spot cleaning or aerosol degreasing in any particular category of workplace, rather it simply recites how OSHA's authority does not extend to self-employed

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[72] 162 Cong. Rec. H3028 (May 24, 2016).

[73] 122 Cong. Rec. H11344 (Sept. 28, 1976).

[74] 50 Fed. Reg. 27674 (July 5, 1985).

[75] Memorandum to Lee M. Thomas from Gerald H. Yamada, June 7, 1985, p. 2.

workers, military personnel, and consumer uses. But those are limitations that were imposed by Congress and have existed since the Occupational Safety and Health Act was enacted (six years before enactment of TSCA). Those limitations apply to every use of every toxic substance. Congress cannot have meant, in enacting "gap-filling" legislation, to open the door to EPA assuming all authority over the use of hazardous substances in the workplace.

If EPA were to identify a category of exposure deemed to present a risk that is unreasonable, these considerations indicate that referral under § 9(a) would be the appropriate course.[76] It is clear from Section 9(a) that TSCA is to be used only when other statutes fail to provide a remedy for unreasonable risks.

Attachments:

Appendix 1
Appendix 2
Appendix 3
Appendix 4
Appendix 5

[76] As noted above, TSCA § 9(a) provides that if the Administrator has reasonable basis to conclude that an unreasonable risk of injury is presented, and he determines, in his discretion, that the risk may be prevented or sufficiently reduced by action under another federal statute not administered by EPA, then the Administrator shall submit a report to that agency describing the risk. In the report, the Administrator shall request that the agency determine if the risk can be prevented or sufficiently reduced by action under the law administered by that agency; if so, the other agency is to issue an order declaring whether the risk described in the Administrator's report is presented, and is to respond to the Administrator regarding its prevention or reduction. The Administrator may set a time (of not less than 90 days) within which the response is to be made. The other agency must publish its response in the Federal Register. If the other agency decides that the risk described is not presented, or within 90 days of publication in the Federal Register initiates action to protect against the risk, EPA may not take any action under § 6 of TSCA.

BEFORE THE ENVIRONMENTAL PROTECTION AGENCY

Trichloroethylene; Regulation of Certain Uses under TSCA § 6(a)

[EPA-HQ-OPPT-2016-0163; FRL-9949-86]

Comments of the

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March 16, 2017

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The Halogenated Solvents Industry Alliance, Inc. (HSIA) represents producers and users of trichloroethylene (TCE). We offer these comments on EPA's proposed rule banning manufacture of TCE for and use of TCE in aerosol degreasing and in spot cleaning by dry cleaning facilities. 81 Fed. Reg. 91592 (Dec. 16, 2016). This rule, proposed under § 6(a) of the Toxic Substances Control Act (TSCA), is based on a Work Plan Assessment of TCE completed by EPA in June 2014. TSCA was amended in June 2016 by the Frank R. Lautenberg Chemical Safety for the 21st Century Act ("Lautenberg Act").

HSIA urges EPA to withdraw the proposed rule, which is based on a very deficient risk assessment.. While EPA is authorized under TSCA § 26(l)(4) to propose a § 6 rule based on a risk assessment completed before TSCA was revised, there is no requirement or deadline for it to do so. The situation is very different for the ten priority compounds designated by EPA under TSCA § 6(b)(2)(A) in December 2016.[1] For these ten designated pollutants, TSCA establishes deadlines for risk assessments and rulemakings. TCE is one of the ten priority compounds, and the better course would be to assess the risks from spot cleaning and aerosol degreasing as part of the required upcoming TCE assessment.

These comments address the following subjects, among others, in detail:

- TSCA § 26(l)(4) requires, for a rule based on a risk assessment completed before TSCA was revised, that the rule must be consistent with "the scope of the completed risk assessment for the chemical substance and consistent with other applicable requirements of § 6." "Although the use of TCE as a solvent degreaser at large commercial/industrial operations" was "not considered in this assessment," EPA nevertheless would prohibit all "commercial use of TCE in aerosol degreasing products," regardless of the size of the facility. This is plainly outside "the scope of the completed risk assessment."

- Further, the TCE Work Plan Assessment does not comply with the requirements of TSCA § 6(b)(4)(F) or TSCA § 26(h) and (i), which are expressly applicable to any EPA "decision based on science" under TSCA § 6. The disparity between the completed risk assessments and the "applicable requirements of § 6" is obvious from even a cursory review of the procedures for risk evaluation under the amended TSCA proposed by EPA earlier this year.

- The Work Plan Assessment expressly relied on hazard values derived directly from a University of Arizona study to estimate non-cancer risk. Several other studies, including two GLP-compliant studies conducted under EPA and OECD guidelines, have been unable to reproduce the effect seen in the Arizona study. The Arizona study has been heavily criticized in the published literature, its results have not been replicated by any other laboratory, and other regulatory authorities (including the California EPA) have rejected the study as deficient.

- Equally, the Work Plan Assessment relies on qualitative and quantitative estimates of cancer risk that are not realistic or justified by any underlying science. EPA estimates a baseline cancer risk from chronic occupational spot cleaning exposures of 1 in 10. Cancer incidence of this magnitude could not go unnoticed, and indeed EPA's estimate is belied by available epidemiology studies of dry cleaning workers which show no such risk. Indeed, two recent large Nordic epidemiological studies, both of which had extensive follow-up of the cohorts, have failed to find an association between TCE and kidney cancer, and these are not addressed in the Work Plan Assessment. Further, EPA's development of a potency factor based on Charbotel *et al.* (2006) directly contravenes the advice EPA received from the National Academy of Sciences.

[1] 81 Fed. Reg. 91927 (Dec.19, 2016).

- On the exposure side, for spot cleaning EPA relied solely on a 2007 California study, which it recognized may not be representative of US dry cleaning facilities. For aerosol degreasing EPA provided no emissions or monitoring data – thus these are hypothetical exposures. Moreover, the draft TCE assessment, entitled "Degreaser and Arts/Crafts Uses," did not address spot cleaning (except to say that none of those sold to consumers contained TCE), but the final Work Plan Assessment is entitled "Degreasing, *Spot Cleaning* and Arts & Crafts Uses" and includes commercial use of TCE as a spotting agent at dry cleaning facilities.

- Because there was no notice that EPA was addressing spot cleaning, there was no participation by dry cleaner representatives and no peer review of the spot cleaning assessment. Moreover, there was no Small Business Advocacy Review, even though spot cleaning is done by dry cleaners which are virtually all small entities. It is not credible that EPA could certify that the rule would not have a significant economic impact on a substantial number of small entities (SISNOSE), where the dry cleaning industry estimates that 60-90% of retail dry cleaners routinely use TCE on the spotting board (14,130 – 21,195 small businesses) and projects that such a ban will cost 4-5% of gross sales, far more than the 1-3% impact considered SISNOSE.

- Peer review of the draft Work Plan Assessment was scathing. Reliance on the unreproducible Arizona study was harshly criticized. The Chair of the panel noted that it was a screening level assessment, not suitable for use in regulation: "*the Agency acted prematurely in issuing this (screening level)* assessment for public comment. . . . After listening carefully to the comments and contributions from the other members of the Panel, I have concluded that there would *little benefit in revising this draft screening assessment.*"[2] Yet EPA claims the peer review was supportive.

- EPA's determination that TCE use in spot cleaning and aerosol degreasing poses an "unreasonable risk" is based on its assessment of risks to workers. It is clear, however, that TSCA is to be used only when other statutes fail to provide a remedy for unreasonable risks. Worker health and safety fall under the jurisdiction of the Occupational Safety and Health Administration (OSHA), and use of TCE in spot cleaning and spray degreasing is already adequately regulated under the Occupational Safety and Health Act. Congress cannot have meant, in enacting "gap-filling" legislation, to open the door to EPA assuming all authority over the use of hazardous substances in the workplace.

I. Failure of Work Plan Assessment to Comply with TSCA §§ 6, 26

A. Applicable Requirements of TSCA §§ 6, 26

Although the Lautenberg Act made significant changes to TSCA to ensure that EPA would employ the "best available science" in its risk assessments, EPA proposes to rely on a remarkably sketchy and inadequate assessment in its inaugural rulemaking under TSCA § 6. TSCA § 6(b)(4)(F), as revised by the Lautenberg Act, requires that EPA's risk evaluations must, among other things:

- "integrate and assess available information on hazards and exposures for the conditions of use of the chemical substance, including information that is relevant to specific risks of injury to health or the environment and information on potentially exposed or susceptible subpopulations identified as relevant by the Administrator;"

[2] https://www.epa.gov/sites/production/files/2015-09/documents/tce_consolidated_peer_review_comments_september_5_2013.pdf.

- "take into account, where relevant, the likely duration, intensity, frequency, and number of exposures under the conditions of use of the chemical substance;" and

- "describe the weight of the scientific evidence for the identified hazard and exposure."

New TSCA § 26(h) requires that, in carrying out § 6, "to the extent that the Administrator makes a decision based on science, the Administrator shall use scientific information, technical procedures, measures, methods, protocols, methodologies, or models, employed in a manner consistent with the best available science, and shall consider as applicable—

> (1) the extent to which the scientific information, technical procedures, measures, methods, protocols, methodologies, or models employed to generate the information are reasonable for and consistent with the intended use of the information;
>
> (2) the extent to which the information is relevant for the Administrator's use in making a decision about a chemical substance or mixture;
>
> (3) the degree of clarity and completeness with which the data, assumptions, methods, quality assurance, and analyses employed to generate the information are documented;
>
> (4) the extent to which the variability and uncertainty in the information, or in the procedures, measures, methods, protocols, methodologies, or models, are evaluated and characterized; and
>
> (5) the extent of independent verification or peer review of the information or of the procedures, measures, methods, protocols, methodologies, or models."

With regard to risk assessments completed prior to passage of the Lautenberg Act, including that for TCE, TSCA § 26(l)(4) provides that "the Administrator may publish proposed and final rules under section 6(a) that are consistent with the scope of the completed risk assessment for the chemical substance and consistent with other applicable requirements of section 6." Thus, EPA may base regulation on the pre-enactment risk assessments only to the extent that they comply with the substantive requirements above.

Regrettably, the proposal to ban TCE in aerosol degreasing addresses a broader scope of uses than considered in the Work Plan Assessment. The scope of that assessment is clear: "although the use of TCE as a solvent degreaser at large commercial/industrial operations is expected to be frequent and the concentration of TCE high, human exposures in these settings are expected to be monitored and controlled by Occupational Safety & Health Administration (OSHA); thus, this use is also not considered in this assessment" (p. 27). The Assessment is focused solely on exposure from TCE use as a solvent degreaser in small commercial settings and by consumers.[3] The proposed ban, however, recognizes no such limitation. It would prohibit commercial use of TCE for general aerosol degreasing, as well as its manufacture, processing, and distribution in commerce for this use. Because the proposed rule would ban uses beyond the scope of the underlying Work Plan Assessment, it is not "consistent with the scope of the completed risk assessment" and therefore does not comply with TSCA § 26(l)(4).

[3] *See* Work Plan Assessment at Table 1-1.

Further, the proposed rule does not comply with the requirements of TSCA § 6(b)(4)(F) or TSCA § 26(h) and (i), which are expressly applicable to any EPA "decision based on science" under TSCA § 6. The disparity between the completed TCE Work Plan Assessment and the "applicable requirements of § 6" is obvious from a review of the procedures for risk evaluation under the amended TSCA proposed by EPA earlier this year.[4]

B. Deficiencies of Principal Non-Cancer Study

1. Not Reproducible

The Work Plan Assessment expressly relies on hazard values derived directly from a single academic study to estimate non-cancer risk.[5] Specifically, it states (p. 104):

> "The acute inhalation risk assessment used developmental toxicity data to evaluate the acute risks for the TSCA TCE use scenarios. As indicated previously, EPA's policy supports the use of developmental studies to evaluate the risks of acute exposures. This policy is based on the presumption that a single exposure of a chemical at a critical window of fetal development, as in the case of cardiac development, may produce adverse developmental effects (EPA, 1991).
>
> "After evaluating the developmental toxicity literature of TCE, the TCE IRIS assessment concluded that the fetal heart malformations are the most sensitive developmental toxicity endpoint associated with TCE exposure (EPA, 2011e). Thus, EPA/OPPT based its acute risk assessment on the most health protective endpoint (i.e., fetal cardiac malformations; Johnson et al., 2003) representing the most sensitive human population (i.e., adult women of childbearing age and fetus >16 yrs).
>
> "The acute risk assessment used the PBPK-derived hazard values (HEC_{50}, HEC_{95}, or HEC_{99}) from Johnson et al. (2003) developmental study for each degreaser and spot cleaner use scenario. . . . These extremely low values result in margin of exposure ("MOE") values below 10 for almost all the occupational and residential exposure scenarios examined."

A single flawed study should not be the basis for the toxicological value that serves as the basis for regulation. Several other studies, including three GLP-compliant studies conducted under EPA guidelines to support pesticide registration (40 CFR § 870.3700) and Organization for Economic Coordination & Development ("OECD") guidelines (414) have been unable to reproduce the effect seen by Johnson *et al.* (2003).

Johnson *et al.* (2003) reported cardiac effects in rats from research carried out at the University of Arizona and originally published ten years earlier by the same authors.[6] In the earlier-published study, there was no difference in the percentage of cardiac abnormalities in rats dosed during both pre-mating

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[4] 82 Fed. Reg. 7562 (Jan. 19, 2017).

[5] Johnson PD, *et al.*, Threshold of trichloroethylene contamination in maternal drinking waters affecting fetal heart development in the rat, Environ Health Perspect. 111:289-92 (2003).

[6] Dawson, B, *et al.*, Cardiac teratogenesis of halogenated hydrocarbon-contaminated drinking water, J. Am. Coll. Cardiol. 21: 1466-72 (1993).

and pregnancy at drinking water exposures of 1100 ppm (9.2%) and 1.5 ppm (8.2%), even though there was a 733-fold difference in the concentrations. The authors reported that the effects seen at these exposures were statistically higher than the percent abnormalities in controls (3%). For animals dosed only during the pregnancy period, the abnormalities in rats dosed at 1100 ppm (10.4%) were statistically higher than at 1.5 ppm (5.5%), but those dosed at 1.5 ppm were not statistically different from the controls. Thus, no meaningful dose-response relationship was observed in either treatment group. Johnson *et al.* republished in 2003 data from the 1.5 and 1100 ppm dose groups published by Dawson *et al.* in 1993 and pooled control data from other studies, an inappropriate statistical practice, to conclude that rats exposed to levels of TCE greater than 250 ppb during pregnancy have increased incidences of cardiac malformations in their fetuses.

2. Criticism in Literature and by Other Regulators

Johnson *et al.* (2003) has been heavily criticized in the published literature.[7] Indeed, its predecessor study was expressly rejected as the basis for MRLs by the Agency for Toxic Substances & Disease Registry (ATSDR) in its last TCE Toxicological Profile Update.[8] Moreover, the Johnson *et al.* (2003) findings were not reproduced in a study designed to detect cardiac malformations; this despite employing an improved method for assessing cardiac defects and the participation of Dr. Johnson herself.[9] No increase in cardiac malformations was observed in the second guideline study,[10] despite high inhalation doses and techniques capable of detecting most of the malformation types reported by Johnson *et al.* (2003). The dose-response relationship reported in Johnson *et al.* (2003) for doses spanning an extreme range of experimental dose levels is considered by many to be improbable, and has not been replicated by any other laboratory.[11]

Even the California Office of Environmental Health Hazard Assessment (OEHHA) rejected the study as deficient:

> "Johnson et al. (2003) reported a dose-related increased incidence of abnormal hearts in offspring of Sprague Dawley rats treated during pregnancy with 0, 2.5 ppb, 250 ppb, 1.5 ppm, and 1,100 ppm TCE in drinking water (0, 0.00045, 0.048, 0.218, and 128.52 mg/kg-day, respectively). The NOAEL for the Johnson study was reported to be 2.5 ppb (0.00045 mg/kg-day) in this short exposure (22 days) study. The percentage of abnormal hearts in the control group was 2.2 percent, and in the treated groups was 0 percent (low dose), 4.5 percent (mid dose 1), 5.0 percent (mid dose 2), and 10.5 percent (high dose).

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[7] Hardin, B, *et al.*, Trichloroethylene and cardiac malformations, Environ. Health Perspect. 112: A607-8 (2004); Watson, R., *et al.*, Trichloroethylene-contaminated drinking water and congenital heart defects: a critical analysis of the literature, Repro. Toxicol. 21: 117-47 (2006).

[8] ATSDR concluded that "[t]he study is limited in that only two widely spaced exposure concentrations were used and that a significant dose-response was not observed for several exposure scenarios." Toxicological Profile for Trichloroethylene Update (September 1997), at 88.

[9] Fisher, J, *et al.*, Trichloroethylene, trichloroacetic acid, and dichloroacetic acid: do they affect fetal rat heart development? Int. J. Toxicol. 20: 257-67 (2001).

[10] Carney, E, *et al.*, Developmental toxicity studies in Crl:Cd (SD) rats following inhalation exposure to trichloroethylene and perchloroethylene, Birth Defects Research (Part B) 77: 405–412 (2006).

[11] "Johnson and Dawson, with their collaborators, are alone in reporting that TCE is a 'specific' cardiac teratogen." Hardin, B, *et al.*, Trichloroethylene and cardiac malformations, Environ. Health Perspect. 112: A607-8 (2004).

> The number of litters with fetuses with abnormal hearts was 16.4 percent, 0 percent, 44 percent, 38 percent, and 67 percent for the control, low, mid 1, mid 2, and high dose, respectively. The reported NOAEL is separated by 100-fold from the next higher dose level. The data for this study were not used to calculate a public-health protective concentration since a meaningful or interpretable dose-response relationship was not observed. *These results are also not consistent with earlier developmental and reproductive toxicological studies done outside this lab in mice, rats, and rabbits: The other studies did not find adverse effects on fertility or embryonic development, aside from those associated with maternal toxicity (Hardin et al., 2004).*"[12]

3. Reservations of EPA Scientific Staff

Remarkably, an EPA staff review that was placed in the docket for the Work Plan Assessment reflects similar concerns. First, one staff member dissented over relying at all on the Arizona study:

> "The rodent developmental toxicology studies conducted by Dawson et al. (1993), Johnson et al. (2003), and Johnson et al. (1998) that have reported cardiac defects resulting from TCE (and metabolite) drinking water exposures have study design and reporting limitations. Additionally, two good quality (GLP) inhalation and gavage rodent studies conducted in other laboratories, Carney et al. (2006) and Fisher et al. (2001), respectively, have not detected cardiac defects. These limitations and uncertainties were the basis of the single dissenting opinion of a team member regarding whether the database supports a conclusion that TCE exposures during development are likely to cause cardiac defects."[13]

Second, even the EPA staff that agreed with use of the study had little confidence that it supported the dose-response assessment:

> "[A] majority of the team members agreed that the Johnson et al. (2003) study was suitable for use in deriving a point of departure. However, confidence of team members in the dose response evaluation of the cardiac defect data from the Johnson et al. (2003) study was characterized as between 'low' and 'medium' (with 7 of 11 team members rating confidence as 'low' and four team members rating confidence as 'low to medium')."[14]

It is surprising that EPA would consider use of a dose-response value for regulation from a study in which seven of its own scientists expressed "low" confidence, and in which the other four could muster no more than "low to medium" confidence. The same report notes: "In conclusion, there has not been a confirmation of the results of the Johnson et al. (2003) and Dawson et al. (1993) studies by another laboratory, but there has also not been a repeat of the exact same study design that would corroborate or refute their findings."

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[12] California EPA Public Health Goal for Trichloroethylene in Drinking Water (July 2009), at 21 (emphasis added).

[13] TCE Developmental Cardiac Toxicity Assessment Update (available at http://www.regulations.gov/#!documentDetail;D=EPA-HQ-OPPT-2012-0723-0045).

[14] *Id.*

4. EPA's Dose-Response Evaluation using Johnson *et al.* (2003) Is Inappropriate

The TCE Work Plan Assessment relies on the prior IRIS Assessment's evaluation of the relationship between TCE exposure dose and the development of cardiac defects, as described in Johnson *et al.* (2003). Ignoring for the moment the myriad of methodological deficiencies in the paper, a closer look at EPA's evaluation of that dose-response relationship in generating a point of departure (POD) raises several concerns. The importance of this activity cannot be overstated, as according to a paper published by the authors of the IRIS Assessment, Johnson *et al.* (2003) represents "the only available study potentially useable for dose-response analysis of fetal cardiac defects."[15]

In discussing the dose-response evaluation, Makris *et al.* (2016) further state that "[g]iven the uncertainties in the dose-response analysis related to the nature of the data, the confidence in the POD based on Johnson *et al.* (2003) has limitations. Overall, however, the POD derived in the 2011 TCE assessment (U.S. EPA, 2011), which used an approach consistent with standard U.S. EPA dose-response practices, remains a reasonable choice." It should be noted that, in order to achieve a better model fit in its derivation of a POD, EPA dropped the highest exposure dose from Johnson *et al.* (2003). With already questionable data, and no expectation that the highest dose of TCE would result in a diminished response, that decision should be reconsidered.

Makris *et al.* (2016) describe additional dose-response analyses performed to characterize the uncertainty in the POD. In summarizing the results of this analysis, they state that "[a]lternative PODs were derived based on use of alternative models, alternative BMR levels, or alternative procedures (such as LOAEL/NOAEL approach), each with different strengths and limitations. These alternatives were within *about an order of magnitude of the POD derived in the 2011 TCE assessment*" (emphasis added). This level of uncertainty in modeling the POD when combined with the uncertainty in the PBPK modeling (discussed elsewhere) and the overall poor quality of the underlying developmental toxicity study provide little confidence in the resulting non-cancer toxicological value in the Work Plan Assessment that drives the proposed regulation.

5. Reliance on Johnson *et al.* (2003) Is Inconsistent with Use of Best Available Science

All acute inhalation exposures in the TCE Work Plan Assessment were measured against potential developmental toxicity endpoints based solely on EPA's IRIS evaluation of Johnson *et al.* (2003). When HSIA requested access to the data used by EPA in its evaluation of the dose-response relationship between TCE exposure and cardiac defects reported in Johnson *et al.* (2003), the Agency provided the spreadsheet, referenced as Johnson (2009) (HERO ID 783484) in the 2011 IRIS Assessment, and indicated that was the entirety of the data evaluated. Examination of that spreadsheet reveals an absence of certain critical information, including, most importantly, dates for any of the individual treatment/control animals.

Acknowledging the documented deficiencies in their paper (and the data provided to EPA), the authors published an erratum aimed at updating the public record regarding methodological issues for Johnson *et al.* (2003).[16] According to Makris *et al.* (2016):

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[15] Makris SL, Scott CS, Fox J, *et al.*, Systematic evaluation of the potential effects of trichloroethylene exposure on cardiac development. Repro Toxicol (2016); http://dx.doi.org/10.1016/j.reprotox.2016.08.014

[16] Johnson PD, Goldberg SJ, Mays MZ, Dawson BV, Erratum: Erratum for Johnson *et al.* [Environ Health Perspect 113: A18 (2005)]; Environ Health Perspect 122: A94 (2014); http://dx.doi.org/10.1289/ehp.122-A94

"some study reporting and methodological details remain unknown, *e.g.*, the precise dates that each individual control animal was on study, maternal body weight/food consumption and clinical observation data, and the detailed results of analytical chemistry testing for dose concentration. Additional possible sources of uncertainty identified for these studies include that the research was conducted over a 6-yr period, that combined control data were used for comparison to treated groups, and that exposure characterization may be imprecise because tap (rather than distilled) drinking water was used in the Dawson *et al.* (1993) study and because TCE intake values were derived from water consumption measures of group-housed animals."

HSIA submits that the information contained in the above paragraph alone should disqualify Johnson *et al.* (2003) as "best available science" as required under EPA's proposed procedures for chemical risk evaluation under TSCA as amended.[17]

6. Failure to Conform to EPA Guidelines for Developmental Toxicity Risk Assessment

EPA's Guidelines for Developmental Toxicity Risk Assessment establish the framework for evaluation of developmental toxicity risk on a case-by-case basis.[18] Under these Guidelines, "[i]f data are considered *sufficient* for risk assessment, an oral or dermal reference dose for developmental toxicity (RfD_{DT}) or an inhalation reference concentration for developmental toxicity (RfC_{DT}) is then derived for comparison with human exposure estimates" (emphasis added).

In defining sufficiency, the Guidelines state: "In the case of animal data, agents that have been tested adequately in laboratory animals *according to current test guidelines* generally would be included in the "Sufficient Experimental Animal Evidence/Limited Human Data" category (emphasis added)." Where, as here, the "database on a particular agent includes less than the minimum sufficient evidence (as defined in the 'Insufficient Evidence' category) necessary for a risk assessment, but some data are available, this information could be used to determine the need for additional testing. . . . In some cases, a database may contain conflicting data. In these instances, the risk assessor must consider each study's strengths and weaknesses within the context of the overall database in an attempt to define the strength of evidence of the database for assessing the potential for developmental toxicity."

Given the demonstrated shortcomings of Johnson *et al.* (2003), which was not conducted to EPA test guidelines, and the availability to EPA of three guideline studies, we submit that the Guidelines for Developmental Toxicity Risk Assessment and TSCA §§ 6 and 26 require a weight of evidence evaluation of the database before EPA relies on Johnson *et al.* (2003) for regulatory purposes.

7. New Relevant Information

A third guideline study of TCE developmental toxicity has been sponsored by HSIA. The study was designed with a focus on cardiac abnormalities and included toxicokinetic measures to enable comparison with the earlier studies. It was intended to fill the remaining gap for a guideline study by the drinking water route, the same exposure route as Johnson *et al.* (2003). Regrettably, although the in-life

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[17] 82 Fed. Reg. 7562 (Jan. 19, 2017).

[18] 56 Fed. Reg. 63798 (December 5, 1991).

portion of the study was conducted during October and November, 2016, the concentrations of TCE measured in the drinking water solutions were found to be below the acceptable target range of 100% ± 10%, invalidating the study. The laboratory is conducting additional studies to identify the source of the deviations and the study will be rerun as soon as the dosing methodological issues are resolved and scheduling permits. A statement to this effect is attached as Appendix 1.

C. Deficiencies of Cancer Risk Assessment

1. Erroneous Characterization of TCE as "Carcinogenic to Humans"

While acute risks of developmental toxicity are characterized by EPA as of the greatest concern, the Work Plan Assessment also concludes that all but one of the degreaser exposure scenarios exceeded all the target cancer levels. The discussion of carcinogenicity in the Work Plan Assessment suffers from slavish reliance on EPA's earlier assessment of TCE under its Integrated Risk Information System.[19] The IRIS Assessment classifies TCE as "Carcinogenic to Humans." It fails to discuss (or even to recognize) that such classification is inconsistent with a definitive report by the National Academy of Sciences, discussed below.[20] We briefly address below how the epidemiological data on TCE do not meet the threshold for classification as "Carcinogenic to Humans."

a. Guidelines for Carcinogen Risk Assessment

EPA's 2005 Guidelines for Carcinogen Risk Assessment provide the following descriptors as to the weight of evidence for carcinogenicity:

- Carcinogenic to humans,
- Likely to be carcinogenic to humans,
- Suggestive evidence of carcinogenicity,
- Inadequate information to assess carcinogenic potential, and
- Not likely to be carcinogenic to humans.[21]

According to the Guidelines, "carcinogenic to humans" means the following:

"This descriptor indicates strong evidence of human carcinogenicity. It covers different combinations of evidence.

- "This descriptor is appropriate when there is convincing epidemiologic evidence of a causal association between human exposure and cancer.

[19] "Toxicological Review of Trichloroethylene (CAS No. 79-01-6) in Support of Summary Information on the Integrated Risk Information System (IRIS)" ("IRIS Assessment")

[20] National Research Council, Contaminated Water Supplies at Camp Lejeune: Assessing Potential Health Effects (2009) (hereinafter "Camp Lejeune report").

[21] 70 Fed. Reg. 17766-817 (April 7, 2005).

- "Exceptionally, this descriptor may be equally appropriate with a lesser weight of epidemiologic evidence that is strengthened by other lines of evidence. It can be used when *all* of the following conditions are met: (a) There is strong evidence of an association between human exposure and either cancer or the key precursor events of the agent's mode of action but not enough for a causal association, *and* (b) there is extensive evidence of carcinogenicity in animals, *and* (c) the mode(s) of carcinogenic action and associated key precursor events have been identified in animals, *and* (d) there is strong evidence that the key precursor events that precede the cancer response in animals are anticipated to occur in humans and progress to tumors, based on available biological information. In this case, the narrative includes a summary of both the experimental and epidemiologic information on mode of action and also an indication of the relative weight that each source of information carries, *e.g.*, based on human information, based on limited human and extensive animal experiments."

According to the Guidelines, the descriptor "likely to be carcinogenic to humans":

"is appropriate when the weight of the evidence is adequate to demonstrate carcinogenic potential to humans but does not reach the weight of evidence for the descriptor 'Carcinogenic to Humans.' Adequate evidence consistent with this descriptor covers a broad spectrum. . . . Supporting data for this descriptor may include:

"An agent demonstrating a plausible (but not definitively causal) association between human exposure and cancer:

- "An agent that has tested positive in animal experiments in more than one species, sex, strain, site or exposure route, with or without evidence of carcinogenicity in humans;

- "A positive tumor study that raises additional biological concerns beyond that of a statistically significant result, for example, a high degree of malignancy or an early age at onset;

- "A rare animal tumor response in a single experiment that is assumed to be relevant to humans; or

- "A positive tumor study that is strengthened by other lines of evidence."

According to the Guidelines, the descriptor "suggestive evidence of carcinogenicity":

"is appropriate when the weight of evidence is suggestive of carcinogenicity; a concern for potential carcinogenic effects in humans is raised, but the data are judged not sufficient for a stronger conclusion. This descriptor covers a spectrum of evidence associated with varying levels of concern for carcinogenicity, ranging from a positive cancer result in the only study on an agent to a single positive cancer result in an extensive database that includes negative studies in other species. Depending on the extent of the database, additional studies may or may not provide further insights. Some examples include:

- "A small, and possibly not statistically significant, increase in tumor incidence observed in a single animal or human study that does not reach the weight of evidence for the descriptor 'Likely to Be Carcinogenic to Humans;'

- "A small increase in a tumor with a high background rate in that sex and strain, when there is some but insufficient evidence that the observed tumors may be due to intrinsic factors that cause background tumors and not due to the agent being assessed;

- "Evidence of a positive response in a study whose power, design, or conduct limits the ability to draw a confident conclusion (but does not make the study fatally flawed), but where the carcinogenic potential is strengthened by other lines of evidence; or

- "A statistically significant increase at one dose only, but no significant response at the other doses and no overall trend."

b. Application of the Guidelines to TCE

In considering the data in the context of applying the "Carcinogenic to Humans" descriptor, one first considers the weight of the epidemiological evidence. We judge the epidemiologic evidence to be neither "convincing" nor "strong," two key terms in the Guidelines. This judgment is based on four recent reviews and meta-analyses of occupational TCE exposures and cancer as well as other reviews of this literature.[22] The recent review and meta-analysis by Kelsh *et al.* focuses on occupational TCE exposure and kidney cancer, and includes the Charbotel *et al.* study that is emphasized in the IRIS and Work Plan Assessments.[23] Both the EPA meta-analysis and the Kelsh *et al.* meta-analysis of the TCE kidney cancer epidemiologic literature produced similar summary results. However in Kelsh *et al.* the limitations of this body of research, namely exposure assessment limitations, potential unmeasured confounding, potential selection biases, and inconsistent findings across groups of studies, did not allow for a conclusion that there is sufficient evidence of a causal association, despite a modest overall association.

There are reasonably well-designed and well-conducted epidemiologic studies that report no association between TCE and cancer, some reasonably well-designed and conducted studies that did report associations between TCE and cancer, and finally some relatively poorly designed studies reporting both positive and negative findings. Overall, the summary relative risks or odds ratios in the meta-analysis studies (EPA or published meta-analyses) generally ranged between 1.2 and 1.4. The draft assessment refers to these associations as "small," a term not

[22] Alexander, D, *et al.*, A meta-analysis of occupational trichloroethylene exposure and multiple myeloma or leukaemia, Occup Med (Lond) 56:485–493 (2006); Alexander, D, *et al.*, A meta-analysis of occupational trichloroethylene exposure and liver cancer, Int Arch Occup Environ Health 81(2):127–43 (2007); Mandel, J, *et al.*, Occupational trichloroethylene exposure and non-Hodgkin's lymphoma: a meta-analysis and review, Occup Environ Med 63:597–607 (2006); Kelsh, M, *et al.*, Occupational trichloroethylene exposure and kidney cancer: a meta-analysis, Epidemiology 21(1): 95-102 (January 2010).

[23] Charbotel, B, *et al.*, Case-control study on renal cell cancer and occupational exposure to trichloroethylene, Part II: Epidemiological aspects, Ann Occup Hyg 50(8):777–787 (2006).

typically consistent with "convincing" and "strong." Weak or small associations may be more likely to be influenced by or be the result of confounding or bias. Smoking and body mass index are well-established risk factors for kidney cancer, and smoking and alcohol are risk factors for liver cancer, yet the potential impact of these factors on the meta-analysis associations was not fully considered. There were suggestions that these factors may have impacted findings (*e.g.*, in the large Danish cohort study of TCE exposed workers, the researchers noted that smoking was more prevalent among the TCE exposed populations, however little empirical data were provided). In addition, co-linearity of occupational exposures (*i.e.*, TCE exposure correlated with chemical and/or other exposures) may make it difficult to isolate potential effects of TCE from those of other exposures within a given study, and hinder interpretation across studies. For example, although Charbotel *et al.* reported potential exposure response trends, while controlling for many confounders of concern (which strengthens the weight of evidence), they also reported attenuated associations for cumulative TCE exposure after adjustment for exposure to cutting fluids and other petroleum oils (weakening the weight of the evidence). This study is also limited due to other potential study design considerations such as selection bias, self reporting of work histories, and residual confounding.

When examining the data for TCE and non-Hodgkin lymphoma, kidney cancer, and liver cancer, associations were inconsistent across occupational groups (summary results differed between aerospace/aircraft worker cohorts compared with workers from other industries), study design, location of the study, quality of exposure assessment (*e.g.*, evaluating studies that relied upon biomonitoring to estimate exposure *vs.* semi-quantitative estimates *vs.* self-report, etc.), and by incidence *vs.* mortality endpoints. Although EPA examined high dose categories, it did not evaluate any potential dose-response relationships across the epidemiologic studies (except for Charbotel *et al.*). Reviews of the epidemiologic data reported in various studies for different exposure levels (*e.g.*, cumulative exposure and duration of exposure metrics) did not find consistent dose-response associations between TCE and the three cancer sites under review.[24] An established dose-response trend is one of the more important factors when making assessments of causation in epidemiologic literature. Thus, based on an overall weight of evidence analysis of the epidemiologic research, these data do not support the conclusion that there is "strong" or "convincing" evidence of a causal association between human exposure and cancer.

EPA's Guidelines also state that a chemical may be described as "Carcinogenic to Humans" with a lesser weight of epidemiologic evidence that is strengthened by other lines of evidence, all of which must be met. One of these lines of evidence is "extensive evidence of carcinogenicity in animals." Therefore, we must briefly evaluate the animal data.

The criteria that have to be met for animal data to support a "carcinogenic to humans" classification are stated in a sequential manner with an emphasized requirement that all criteria have to be met. Since the Guidelines consider this to be an "exceptional" route to a "carcinogenic to humans" classification, we would expect rigor to have been applied in assessing animal data against the criteria. This simply was not done.

Of the four primary tissues that EPA evaluated for carcinogenicity, only one or perhaps two rise to the level of biological significance. Discussion of the remaining tumor types appears to presuppose

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[24] Mandel, J, *et al.*, Occupational trichloroethylene exposure and non-Hodgkin's lymphoma: a meta-analysis and review, Occup Environ Med 63:597–607 (2006); Alexander, D, *et al.*, A meta-analysis of occupational trichloroethylene exposure and liver cancer, Int Arch Occup Environ Health 81(2):127–43 (2007); Kelsh, M, *et al.*, Occupational trichloroethylene exposure and kidney cancer: a meta-analysis, Epidemiology 21(1): 95-102 (January 2010).

that TCE is carcinogenic. The resulting discussion appears then to overly discount negative data, of which there are many, and to highlight marginal findings. The text does not appear to be a dispassionate rendering of the available data. Specifically, EPA's conclusion that kidney cancer is evident in rats rests on *one* statistically significant finding in over 70 dose/tumor endpoint comparisons and references to exceedances of historical control values.[25] Using a 0.05 p-value for statistical significance, a frequency of 1 or even several statistically or biologically significant events is expected in such a large number of dosed/tumor groups. EPA's overall conclusion based on these flawed studies cannot be that TCE is a known kidney tumorigen. The best that can be said is that the data are inconsistent. Certainly they do not meet the criterion of "extensive evidence of carcinogenicity in animals." Several marginal findings do not constitute "extensive evidence."

For all these reasons, EPA's classification of TCE as "Carcinogenic to Humans" is not supported by the evidence and cannot be justified under the 2005 Guidelines.[26]

c. EPA's Position that there is 'Convincing Evidence' that TCE Is Carcinogenic to Humans is Inconsistent with National Academy of Sciences Conclusion of only 'Limited or Suggestive Evidence'

The IRIS Assessment states that "TCE is characterized as 'carcinogenic to humans' by all routes of exposure. This conclusion is based on convincing evidence of a causal association between TCE exposure in humans and kidney cancer."

Box 2 of the Academy's Camp Lejeune report, attached as Appendix 3, categorizes every cancer outcome reviewed in relation to exposure to TCE, the dry cleaning solvent perchloroethylene, or a mixture of the two. The categories are taken directly from a respected Institute of Medicine (IOM) report.[27] These categories are "sufficient evidence of a causal relationship," "sufficient evidence of an association," "limited or suggestive evidence of an association," "inadequate evidence to determine an association," and "limited or suggestive evidence of no association," all as defined in Box 1, also attached.

Looking at Box 2, evidence considered by EPA to be "convincing evidence of a causal association between TCE exposure in humans and kidney cancer" would seem to be considered "sufficient evidence of a causal relationship." Yet the Academy found no outcomes in that category. It would at least be "sufficient evidence of an association." Again, the Academy found no outcomes in that category. Only in the third category, "limited or suggestive evidence of an association," does one find kidney or any other cancer outcome associated with TCE.

"Limited evidence of an association" is far from "convincing evidence of causation." One would expect at the least a detailed explanation of EPA's very different conclusion. Although the 2009 Camp Lejeune study was already published, and indeed is cited in the references, there is no mention of it in the text of the IRIS Assessment, even though the previous draft had just been the subject of a multi-year review by the Academy.

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[25] And that bioassay is from a laboratory whose studies EPA has reviewed and declined to rely upon in other assessments.

[26] Further commentary to this effect, provided by a distinguished group of consultants in connection with the TCE IRIS Assessment but not addressed by EPA, is attached as Appendix 2.

[27] Institute of Medicine, Gulf War and Health, Vol. 2, Insecticides and Solvents (National Academies Press) (2003).

The Camp Lejeune committee began with a comprehensive review of the epidemiology studies of the two solvents by the IOM for its Gulf War Report. They then identified new studies published from 2003 to 2008 and considered whether these changed the conclusions in the IOM report. In the case of TCE and kidney cancer, this was the case. The Camp Lejeune committee considered six new cohort studies and two case-control studies (including Charbotel *et al.*). They concluded that several of these studies reported an increased risk of kidney cancer, but observed that the results were often based on a relatively small number of exposed persons and varied quality of exposure data and methodology. Given these data, the committee raised the classification for TCE to match the IOM conclusion of "limited" evidence for perchloroethylene.

EPA, on the other hand, offered the summary conclusion of convincing human evidence, based on the "consistency" of increased kidney cancer across the different studies. The authors of these studies, however, do not agree with EPA's characterization of them. For example, the authors of Charbotel *et al.*, the study EPA finds most compelling, state that the "study suggests an association between exposures to high levels of TCE and increased risk of [renal cell carcinoma]. Further epidemiological studies are necessary to analyze the effect of lower levels of exposure."

Given the flaws in the IRIS Assessment, and the very different conclusion reached by the Academy in its Camp Lejeune report on the same body of data, the Work Plan Assessment should not rely on the IRIS Assessment's classification of TCE as "Carcinogenic to Humans."

2. EPA Should Reassess Available Cancer Epidemiology Data, Given Publication of More Recent and Larger Studies on Worker Populations

The observation of an elevated but weak kidney cancer association reported by Charbotel *et al.* (2006)[28] contrasts with other occupational studies which did not find an elevation in kidney cancer in industries using TCE as a metal degreaser, *e.g.*, aircraft manufacturing, metal cleaning, etc., where exposures may be higher than for screw cutters. Lipworth and coworkers (2011) found no evidence of increased kidney cancer in a large worker cohort with multiple decades of TCE exposure and extended cancer follow-up evaluations.[29] The aircraft manufacturing study involved a total cohort of 77,943 workers, of which 5,443 were identified as exposed to TCE. The study involved evaluations from 1960 through 2008, at which time 34,248 workers had died. Approximately 30% of the workers were hired before 1960 (60% born before 1940), 52% terminated employment by 1980, and approximately a third of the workers were employed for more than 20 years. The standardized incidence ratio (SIR) for kidney cancer in the TCE-exposed workers was reported as 0.66 (CI 95%: 0.38-1.07). This value for the SIR indicates that these workers were potentially less likely to get kidney cancer than the normal population (or at least had the same rate as the normal population – SIR of 1).

More recently, two large Nordic country epidemiological studies, both of which had extensive follow-up of the cohorts, have likewise failed to find an association between TCE and kidney cancer. An SIR of 1.01 (0.70-1.42) was found by Hansen *et al.* (2013) for kidney cancer based on 32 cases out of a total of 997 cancer cases in a cohort of 5,553 workers in Finland, Sweden, and Denmark, indicating that

[28] Charbotel, B, *et al.*, Case-control study on renal cell cancer and occupational exposure to trichloroethylene, Part II: Epidemiological aspects, Ann Occup Hyg 50(8):777–787 (2006).

[29] Lipworth L, Sonderman JS, Mumma MT, *et al.*, Cancer mortality among aircraft manufacturing workers: an extended follow-up, J Occup Environ Med 53(9): 992-1007 (2011).

rates were the same as the normal population.[30] TCE exposures in this cohort were directly confirmed from urinary biomonitoring of the TCE metabolite trichloroacetic acid (TCA). However, overall TCE exposures were likely low in this cohort in that most urinary TCA measurements were less than 50 mg/L, corresponding to approximately 20 ppm TCE exposure. Thus, consistent with the conclusions of Bruning *et al.* (2003),[31] this study indicates TCE is unlikely to be a low-dose kidney carcinogen.

Similarly, no evidence of kidney cancer was found by Vlaanderen *et al.* (2013) in a recent follow-up examination of the Nordic Occupational Cancer cohort (Finland, Iceland, Norway, Sweden) in which statistically non-significant risk ratios (RR) of 1.01 (0.95-1.07), 1.02 (0.97-1.08), and 1.00 (0.95-1.07) were reported for a total of 4,145 renal cancer cases approximately equally distributed across three respective TCE exposure groups (tertiles) assigned from a job exposure matrix analysis.[32] Finally, although a meta-analysis of 23 studies meeting criteria for study inclusion found a slightly increased simple summary association of TCE and kidney cancer, RR 1.42 (1.17-1.77), more detailed analyses of subgroups suggested no association, or possibly a moderate elevation in kidney cancer risk, and no evidence of increasing risk with increasing exposure.[33]

These more recent studies were not reviewed in the 2011 TCE IRIS Assessment or the 2014 TCE Work Plan Assessment that form the basis for the proposed regulation. Any regulatory action under TSCA § 6, however, is required to be based on the "best available science" supported by "substantial evidence in the record." This provides compelling support for our position that the instant proposal should be withdrawn and the uses under consideration be examined following the TCE assessment EPA will be conducting in the near future under TSCA § 6(b)(4)(A).

3. EPA's Reliance on Charbotel *et al.* (2006) Results in an Overly Conservative Estimate of Risk

In its 2014 Work Plan Assessment of potential cancer risk, EPA focused solely on inhalation exposures and relied on an inhalation unit risk (IUR) value developed in the 2011 IRIS Assessment. The IUR was based primarily on epidemiology data from the case-control study on renal cell carcinoma (RCC) by Charbotel *et al.* (2006), discussed above. Although other epidemiological studies were used to derive an adjusted IUR estimate for the combined risk of developing RCC, NHL, or liver cancer, EPA concedes a lower level of confidence in both the NHL and liver cancer databases. While the Charbotel *et al.* study suggests a relationship between cumulative TCE exposure and RCC incidence, the reliability of the exposure estimates is a major concern.

The National Academy of Sciences Committee that reviewed the draft IRIS assessment released in 2001 recommended that:

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[30] Hansen J, Sallmén M, Seldén AI, *et al.*, Risk of cancer among workers exposed to trichloroethylene: analysis of three Nordic cohort studies, J Natl Cancer Inst 105(12): 869-877 (2013).

[31] Brüning T, Pesch B, Wiesenhütter B, *et al.*, Renal cell cancer risk and occupational exposure to trichloroethylene: Results of a consecutive case-control study in Arnsberg, Germany, Am J Ind Med. 43(3): 274-285 (2003).

[32] Vlaanderen J, Straif K, Pukkala E, *et al.*, Occupational exposure to trichloroethylene and perchloroethylene and the risk of lymphoma, liver, and kidney cancer in four Nordic countries, Occup Environ Med 70(6): 393-401 (2013).

[33] Kelsh MA, Alexander DD, Mink PJ, Mandel JH, Occupational trichloroethylene exposure and kidney cancer: a meta-analysis, Epidemiology 21(1): 95-102 (2010).

> "[t]here appear to be insufficient epidemiologic data to support quantitative dose-response modeling for trichloroethylene and cancer. The committee recommends that toxicologic data be used to fit the primary dose-response model(s) and that the available epidemiologic data be used only for validation. The committee does not believe that the available information is sufficient to determine the best dose-response model for trichloroethylene."[34]

EPA should follow the recommendation of the National Academy of Sciences, which referenced the Charbotel *et al.* (2005) final study report in its review of TCE.[35] The authors' own conclusions that the study only "suggests that there is a weak association between exposures to TRI [TCE] and increased risk of RCC" argues against the existence of the robust relationship which should be required for a dose-response assessment used as the basis for regulation.[36]

The exposure assessment for the Charbotel study was based on questionnaires and expert judgment, not direct measures of exposure.[37] Worker exposure data from deceased individuals were included in the study. In contrast to living workers, who were able to respond to the questionnaires themselves, exposure information from deceased workers (22.1% of cases and 2.2% of controls) was provided by surviving family members. The authors acknowledge that "this may have led to a misclassification for exposure to TCE due to the lower levels in the quality of information collected."

Analysis of the data revealed evidence of confounding from cutting fluid exposure. Unfortunately, TCE and cutting oil were co-exposures that could not be disaggregated and the majority of

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[34] National Research Council, Assessing the human health risks of trichloroethylene: key scientific issues, National Academies Press, Washington, DC (2006); http://www.nap.edu/openbook.php?record_id=11707&page=R1.

[35] Charbotel B, Fevotte J, Hours M, *et al.*, Case-control study on renal cell cancer and occupational trichloroethylene exposure, in the Arve Valley (France), Lyon, France: Institut Universitaire de Médecine du Travail, UMRESTTE, Université Claude Bernard (2005); http://hal.archives-ouvertes.fr/docs/00/54/59/80/PDF/charbotel_octobre_05.pdf

[36] This concern was recognized by the European Chemicals Agency (ECHA) in its 2013 Chemical Safety Report on TCE: "[T]here are several concerns with this study that should be taken into consideration when assessing its use in risk assessment and hazard characterization. For example, potential selection bias, the quality of the exposure assessment, and the potential confounding due to other exposures in the work place. With respect to the potential for selection bias, no cancer registry was available for this region to identify all relevant renal cell cancer cases from the target population. Case ascertainment relied on records of local urologists and regional medical centers; therefore, selection bias may be a concern. Given the concerns of the medical community in this region regarding renal cell cancer (RCC) among screw cutting industry workers, it is likely that any cases of renal cell cancer among these workers would likely be diagnosed more accurately and earlier. It is also much more unlikely that an RCC case among these workers would be missed compared to the chance of missing an RCC case among other workers not exposed to TCE. This preference in identifying cases among screw-cutting industry workers would bias findings in an upward direction. Concerning the potential for other exposures that could have contributed to the association, screw-cutting industry workers used a variety of oils and other solvents. Charbotel *et al.* reported lower risks for TCE exposure and renal cell cancer once data were adjusted for cutting oils. In fact, they noted, 'Indeed many patients had been exposed to TCE in screw-cutting workshops, where cutting fluids are widely used, making it difficult to distinguish between cutting oil and TCE effects.' This uncertainty questions the reliability of using data from Charbotel *et al.* since one cannot be certain that the observed correlation between kidney cancer and exposure is due to trichloroethylene."

[37] Fevotte J, Charbotel B, Muller-Beauté P, *et al.*, Case-control study on renal cell cancer and occupational exposure to trichloroethylene, Part I: Exposure assessment, Ann Occup Hyg 50: 765-775 (2006); http://dx.doi.org/10.1093/annhyg/mel040.

the TCE exposed population, the screw cutters, could be expected to experience similar patterns of exposure for both TCE and cutting fluids (probably in aerosol form). Thus the apparent dose-response relationship for TCE could be wholly or in part the result of exposure to cutting fluids.

In their 2006 publication of the study results, the authors assigned cumulative exposures into tertiles (i.e., low, medium and high), yet the dose-response evaluation conducted as part of the IRIS Assessment relied on mean cumulative exposure levels provided at a later date.[38] Although the IRIS Assessment references the email submission of the data to EPA, it provides no detail on the technical basis for the table, raising serious transparency issues.

In an apparent acknowledgement of the uncertainty of the exposure information, Charbotel *et al.* (2006) included an evaluation of "the impact of including deceased patients (proxy interviews) and elderly patients (>80 years of age)" on the relationship between exposure to TCE and RCC. Interestingly, it was stated that "only job periods with a high level of confidence with respect to TCE exposure were considered" in the study, an apparent reference to the use of two different occupational questionnaires, one "devoted to the screw-cutting industry and a general one for other jobs." As the Adjusted Odds Ratio (OR) for the high cumulative dose group was actually higher in the censored subgroup than in the uncensored group [3.34 (1.27-8.74) vs 2.16 (1.02-4.60)], the authors cavalierly suggested that "misclassification bias may have led to an underestimation of the risk."

What the authors and EPA appear to have overlooked is that, in addressing the misclassification bias, Charbotel may also have altered the cumulative dose-response relationship. For example, in the censored subgroup there were now only 16 exposed cases (1 in the Low Group, 4 in the Medium Group and 11 in the High Group) with Adjusted ORs of 0.85, 1.03 and 3.34, respectively. If the dose-response relationship in this higher-confidence subgroup has changed, use of the lower-confidence group to calculate the IUR would have to be rigorously justified by EPA before it could be considered sufficiently robust to drive the types of decisions based on unit risk that are found in the proposed rule.

4. Use of TCE Glutathione Conjugate Derived Metabolites Dichlorovinylglutathione (DCVG) and Dichlorovinylcysteine (DCVC) in TCE Renal Toxicity and Cancer Risk Assessment Should Be Reconsidered Given Improved Understanding of the Differential Quantitative Formation of these Metabolites in Animals Relative to the TCE Oxidative Metabolites Trichloroethanol (TCOH), Trichloroacetic Acid (TCA) and Dichloroacetic Acid (DCA)

The TCE IRIS Assessment relies in part on the conclusion that DCVG and DCVC, which are weakly active renal toxicants and genotoxicants, are formed in toxicologically significant concentrations following human exposures to TCE. Importantly, the basis for this conclusion rests on studies in which a relatively high blood DCVG concentration (100 nM) was observed in volunteers exposed for 4 hours to 50 or 100 ppm TCE.[39] However, Lash *et al.* (1999) relied on a colorimetric chromatographic method analysis of TCE glutathione conjugate-derived metabolites which had substantial potential for detection of non-TCE-specific endogenous substances. Subsequent radiochemical and HPLC-MS/MS based analyses that specifically quantitated both DCVG and DCVC have found that the activity of the

[38] Charbotel B (2008) [Email from Barbara Charbotel, University of Lyon, to Cheryl Scott, EPA].

[39] Lash LH, Putt DA, Identification of S-(1,2-dichlorovinyl)glutathione in the blood of human volunteers exposed to trichloroethylene, J Toxicol Env Hlth Part A, 56: 1-21 (1999).

glutathione conjugate pathway is substantially lower than that of the oxidative pathway resulting in TCA and DCA formation in both animals and humans.[40]

Since the publication of the TCE IRIS Assessment in 2011, additional studies have evaluated the kidney concentrations of TCE oxidative and glutathione conjugate-derived metabolites in a variety of mouse strains administered 5 daily oral 600 mg/kg doses of TCE.[41] Metabolites were quantitated 2 hr after the last daily dose in that toxicokinetic evaluations had shown the approximate maximum plasma concentrations of TCA, DCA, DCVG and DCVC were observed 2 hr following oral TCE treatment.[42] Using a structure-specific HPLC-ESI-MS/MS method, Yoo *et al.* (2015) demonstrated that DCVG and DCVC were only a very small fraction of total oxidative metabolites quantitated in kidney. TCOH kidney concentrations were 2-4-fold greater than TCA, and TCA concentrations were 100-1000 greater than DCA. Importantly, DCA concentrations were 100-1000-fold greater than DCVG and DCVC, resulting in the conclusion that TCE oxidative metabolism was up to 5 orders of magnitude greater than glutathione conjugate-derived metabolism. These findings were consistent with the earlier report from Kim *et al.* (2009) in which the plasma toxicokinetics TCA, DCA, DCVG and DCVC following a single 2140 mg/kg oral TCE dose found that the cumulative AUC of oxidative metabolites was 40,000-fold higher than the combined AUC of DCVG and DCVC; note that this study did not quantify TCOH, which would have further increased the disparity of glutathione conjugate-derived relative to oxidative-derived metabolites. These data demonstrate a dramatically lower function glutathione-conjugate metabolism relative to oxidative metabolism in mice, despite the observation by Dekant (2010) that mice generate DCVC at slightly higher rates than rats and greater than 10-fold higher than humans.

The results of studies using structure-specific analytical methods for quantitation of DCVG and DCVC directly challenge the hypothesis that glutathione conjugate-derived metabolites plausibly account for the genotoxicity, renal cytotoxicity, and ultimate carcinogenicity in rodents.[43] DCVC was only marginally cytotoxic (LDH release), if at all, when incubated at 0.2M (200,000 nM) with isolated renal cortical cells of male and female rats. This *in vitro* concentration is substantially higher than the approximate maximum kidney concentrations of 10-75 nM DCVC resulting from treatment of various strains of mice with a high oral TCE dose of 600 mg/kg/day for 5 days observed by Yoo *et al.* (2015). In addition, a likely NOAEL of 1 mg/kg/day was reported for kidney toxicity (no change in serum BUN, weak tubule dilation and no necrosis) in mice administered DCVC orally or intraperitoneally at 1, 10 or 30 mg/kg/day, 1 day per week, for 13 weeks.[44] If, based on Yoo *et al.* (2015), it is assumed that the ratio of formation of oxidative metabolites to glutathione conjugate-derived metabolites is 10,000:1, an implausibly high (occupational or general population) dose of 6044 mg/kg TCE would be required to

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[40] Dekant,W (2010), attached as Appendix 4.

[41] Yoo HS, Bradford BU, Kosyk O, Uehara T, Shymonyak S, Collins LB, Bodnar WM, Ball LM, Gold A, Rusyn I, Comparative analysis of the relationship between trichloroethylene metabolism and tissue-specific toxicity among inbred mouse strains: kidney effects, J Toxicol Env Hlth Pt A, 78: 32-49.b (2015).

[42] Kim, S, Kim, D, Poilack, GM, Collins, LB, and Rusyn, I, Pharmacokinetic analysis of trichloroethylene metabolism in male B6C3F1 mice: Formation and disposition of trichloroacetic acid, dichloroacetic acid, S-(1,2-dichlorovinyl)glutathione and S-(1,2-dichlorovinyl)-L-cysteine, Toxicol Appl Pharmacol 238: 90-99 (2009).

[43] Lash LH, Qian W, Putt DA, Hueni SE, Elfarra AA, Krause RJ, Parker JC, Renal and hepatic toxicity of trichloroethylene and its glutathione-derived metabolites in rats and mice: Sex-, species-, and tissue-dependent differences, J Pharmacol Exp Ther 297: 155-164 (2001).

[44] Shirai N, Ohtsuji M, Hagiwara K, Tomisara H, Ohtsuje N, Hirose S, Hagiwara H, Nephrotoxic effect of subchronic exposures to S-(1,2-dichlorovinyl)-L-cysteine in mice, J Toxicol Sci 37: 871-878.h (2012).

deliver a NOAEL dose of 1 mg/kg/day DCVC (1 mmol/kg/day TCE results in 0.0001 mmol/kg/day DCVC; 1 mg/kg/day DCVC = 0.0046 mmol/kg/day). These dose-toxicity calculations suggest that it appears toxicologically implausible that real-world exposures to TCE are capable of producing doses of DCVC sufficient to cause renal toxicity and carcinogenicity in mice.

D. Peer Review Ignored

The draft Work Plan Assessment was the subject of peer review by a panel selected by EPA in 2013. The peer review report highlights that it was a screening level assessment that inappropriately relied on an unreproducible study, and recommended that the assessment be abandoned.[45] One reviewer devoted six pages to a very detailed critique of Johnson *et al.* (2003) and EPA's reliance on such a deficient study.[46] Nevertheless, EPA ignored the peer review. Remarkably, even though the trade press article on the peer review was entitled *EPA Peer Reviewers Say Trichloroethylene Analysis Not Ready for Regulatory Use*, the EPA Assistant Administrator for Chemical Safety and Pollution Prevention wrote to the EPA Inspector General that "[i]t is notable that *the external peer reviews of all the Work Plan assessments we have completed thus far supported our overall assessment methodologies and conclusions*."[47] A more detailed description of the peer reviewers' comments is attached as Appendix 3.

Ppeer review is identified as a key step in EPA's proposed procedures for chemical risk evaluation under TSCA as amended. EPA states that "[i]n addition to any targeted peer review of specific aspects of the analysis, the entire risk assessment will also undergo peer review, as it is important for peer reviewers to consider how the various underlying analyses fit together to produce an integrated risk characterization which will form the basis of unreasonable risk determination."[48] As the draft Work Plan Assessment for TCE did not address the spot cleaning scenario at all, the assessment of risks under that scenario has *never* been subjected to peer review. Thus an applicable requirement of TSCA §§ 6 and 26(l)(4) for reliance on the Work Plan Assessment has not been met.

E. Screening Level Assessment

As noted above and in Appendix 5, the peer review report highlights that the Work Plan Assessment was a screening level assessment. Specifically, the Chairperson of EPA's peer review panel wrote:

> "The draft document fails to articulate satisfactorily that the analysis described within should be characterized as a screening level assessment. . . . I believe that the Agency acted prematurely in issuing this (screening level) assessment for public comment. . . . After listening carefully to the comments and contributions from the other members of

[45] https://www.epa.gov/sites/production/files/2015-09/documents/tce_consolidated_peer_review_comments_september_5_2013.pdf.

[46] *Id.*

[47] Response to Office of Inspector General Draft Report No. OPE-FY14-0012 "EPA's Risk Assessment Division Has Not Fully Adhered to Its Quality Management Plan," (July 30, 2014), Appendix A, p.10 (available at https://www.epa.gov/sites/production/files/2015-09/documents/20140910-14-p-0350.pdf) (emphasis added). *Compare* BNA Daily Environment Report, *EPA Peer Reviewers Say Trichloroethylene Analysis Not Ready for Regulatory Use* (July 18, 2013).

[48] 82 Fed. Reg. at 7572.

the Panel, I have concluded that there would little benefit in revising this draft screening assessment."

With regard to aerosol degreasing, EPA identified only two aerosol degreasing products containing TCE in the marketplace and found no emissions or monitoring data for either product – thus these are hypothetical exposures. Further, EPA used E-FAST2/CEM modeling to develop "high-end acute inhalation exposure estimates" based solely on professional judgment, providing confirmation that this is a screening level assessment. The highest uncertainties were associated with mass of product used per event, duration of event, and number of events per year, as the values selected were hypothetical, thus leading to further lack of confidence in the assessment.

For spot cleaning workers the problems with the exposure assessment are even more obvious. A major limitation of the exposure assessment used to evaluate potential risk arising from spot cleaning operations was the unavailability of relevant exposure monitoring data. Section 2.4.2.5 of the Work Plan Assessment, however, references a study specific to spot cleaning and states that "site-specific parameters from this study were incorporated into the NF/FF model to obtain site-specific model estimates of worker exposure."[49]

Examination of the NIOSH (1997) study reveals that the air monitoring was actually conducted in response to an OSHA complaint from workers and the report states that "[c]onditions at this shop were probably worst case." Use of monitoring data from a worst case, potential enforcement situation adds additional strength to the concern that the Work Plan Assessment is actually a screening level assessment which does not reflect normal operating conditions and exposures.

It is clear that a risk evaluation that supports a TSCA § 6 rule must be more robust than the screening level Work Plan Assessment that EPA carried out for TCE, which does not comply with Office of Management and Budget ("OMB") guidelines implementing the Information Quality Act.[50] First, EPA must conduct a "highly influential scientific assessment" to support TSCA § 6 rulemaking. OMB defines a scientific assessment as "highly influential" if dissemination of the assessment could have a potential impact of more than $500 million in any one year on either the public or private sector, or if the dissemination is novel, controversial, precedent-setting, or has significant interagency interest.

The TCE assessment employed worst-case or default assumptions that led to overestimation of potential risks. Such assessments may be appropriate to support a decision that no further action or evaluation is necessary, because there is confidence that the potential risks are not a concern. However, they are inappropriate to support regulations intended to reduce risk because screening level assessments do not accurately estimate risk or quantify exposures. Second, OMB's guidelines also require agencies to subject highly influential scientific assessments to more rigorous peer review. For TCE, EPA selected a contractor to manage the peer review process, even though experts consider contractor-managed peer review to be the least rigorous level of peer review.

F. Summary of Concerns

[49] National Institute for Occupational Safety and Health (NIOSH), Control of Health and Safety Hazards in Commercial Dry Cleaning, Publication Number 97-150, Centers for Disease Control and Prevention, Atlanta, GA (1997); http://www.cdc.gov/niosh/docs/97-150/#controls

[50] OMB, Final Information Quality Bulletin for Peer Review (Dec. 16, 2004) (available at https://www.whitehouse.gov/sites/default/files/omb/assets/omb/memoranda/fy2005/m05-03.pdf).

In sum, the TCE Work Plan Assessment is inconsistent with the applicable requirements of revised § 6 in the following ways, among others:

- It expressly relies on hazard values derived directly from a single academic study to estimate non-cancer risk, even though several other studies, including two GLP-compliant studies conducted under EPA guidelines, have been unable to reproduce the effect;[51]

- The University of Arizona study upon which EPA relies has been heavily criticized in the published literature,[52] and other regulatory agencies have expressly declined to rely on the academic study citing data quality concerns;[53]

- The authors of the Arizona study have published repeated corrections that fail to address the data quality concerns;[54] and a majority of EPA's own staff scientists expressed "low" confidence in its results.[55]

- The Work Plan Assessment relies on qualitative and quantitative estimates of cancer risk that are not realistic or justified by any underlying science. Two recent large Nordic epidemiological studies, both of which had extensive follow-up of the cohorts, failed to find an association between TCE and kidney cancer, but these are not addressed in the Work Plan Assessment. Further, EPA's reliance upon a potency factor based on Charbotel *et al.* (2006) directly contravenes the advice EPA received from the National Academy of Sciences

- For aerosol degreasing EPA provided no emissions or monitoring data – thus these are hypothetical exposures. The spot cleaning exposure assessment relies solely on a 2007 California study, which EPA recognized may not be representative of US dry cleaning facilities. The draft TCE Assessment, entitled "Degreaser and Arts/Crafts Uses," did not address spot cleaning at all (except to say that none of those sold to consumers contained TCE), but the final Work Plan Assessment is entitled "Degreasing, *Spot Cleaning* and Arts & Crafts Uses" and includes commercial use of TCE as a spotting agent at dry cleaning facilities.

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[51] *Compare* Johnson *et al.* (2003) to Fisher, J, *et al.*, Trichloroethylene, trichloroacetic acid, and dichloroacetic acid: do they affect fetal rat heart development? Int. J. Toxicol. 20: 257-67 (2001) and Carney, E, *et al.*, Developmental toxicity studies in Crl:Cd (SD) rats following inhalation exposure to trichloroethylene and perchloroethylene, Birth Defects Research (Part B) 77: 405–412 (2006).

[53] *E.g.*, "Johnson and Dawson, with their collaborators, are alone in reporting that TCE is a 'specific' cardiac teratogen." Hardin, B, *et al.*, Trichloroethylene and cardiac malformations, Environ. Health Perspect. 112: A607-8 (2004); Watson, R., *et al.*, Trichloroethylene-contaminated drinking water and congenital heart defects: a critical analysis of the literature, Repro. Toxicol. 21: 117-47 (2006).

[55] *E.g.*, "The data from this study were not used to calculate a public-health protective concentration since a meaningful or interpretable dose-response relationship was not observed. These results are also not consistent with earlier developmental and reproductive toxicological studies done outside this lab in mice, rats, and rabbits." California EPA Public Health Goal for Trichloroethylene in Drinking Water (July 2009), at 21.

[54] Johnson, PD, *et al.*, Environ Health Perspect 122: A94 (2014): erratum to Johnson, PD, *et al.*, Environ Health Perspect 113:A18 (2005), which is an erratum to Johnson *et al.* (2003).

[55] TCE Developmental Cardiac Toxicity Assessment Update (available at http://www.regulations.gov/#!documentDetail;D=EPA-HQ-OPPT-2012-0723-0045).

- It is a screening level assessment which does not meet OMB guidelines implementing the Information Quality Act for a "highly influential scientific assessment" to support TSCA § 6 rulemaking.

- The report of the peer review of the TCE Assessment highlights the foregoing points in the clearest possible terms, but EPA ignored it.[56] In fact, the EPA Assistant Administrator characterized the peer review as supportive.

Following enactment of the Lautenberg Act, it should be clear that a risk evaluation that supports a TSCA § 6 rule must be more robust than the screening level Work Plan Assessment that EPA conducted for TCE. Peer review and public comments identified numerous scientific deficiencies with the draft assessment, including the inappropriate use of default assumptions; ignoring contrary evidence that affects the weight of the scientific evidence; reliance on inapposite exposure data; conclusions inconsistent with the evidence cited; and reliance on a study that is not reproducible. Important shortcomings in both the hazard and exposure assessments were noted. Whatever "best available science" may mean, it cannot include reliance on an unreproducible toxicity study, a cancer risk assessment that does not take into account relevant epidemiological and toxicological studies, or outdated and unrepresentative exposure information.[57] And certainly EPA can no longer afford to ignore the conclusions of the peer review it initiated, as TSCA § 26(h) requires it to consider "the extent of independent verification or peer review of the information."

II. Failure to Comply with SBREFA

The Regulatory Flexibility Act, as amended by the Small Business Regulatory Enforcement Fairness Act (SBREFA), provides:

> "(a) When any rule is promulgated which will have a significant economic impact on a substantial number of small entities, the head of the agency promulgating the rule or the official of the agency with statutory responsibility for the promulgation of the rule shall assure that small entities have been given an opportunity to participate in the rulemaking for the rule through the reasonable use of techniques such as—
>
> (1) the inclusion in an advance notice of proposed rulemaking, if issued, of a statement that the proposed rule may have a significant economic effect on a substantial number of small entities;
>
> (2) the publication of general notice of proposed rulemaking in publications likely to be obtained by small entities;
>
> (3) the direct notification of interested small entities;

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[56] https://www.epa.gov/sites/production/files/2015-09/documents/tce_consolidated_peer_review_comments_september_5_2013.pdf.

[57] *See* 162 Cong. Rec. S3522 (June 7, 2016) ("For far too long Federal agencies have manipulated science to fit predetermined political outcomes, hiding information and underlying data, rather than using open and transparent science to justify fair and objective decision making. This Act seeks to change all of that and ensure that EPA uses the best available science, bases scientific decisions on the weight of the scientific evidence rather than one or two individual cherry-picked studies, and forces a much greater level of transparency that forces EPA to show their work to Congress and the American public.)"

(4) the conduct of open conferences or public hearings concerning the rule for small entities including soliciting and receiving comments over computer networks; and

(5) the adoption or modification of agency procedural rules to reduce the cost or complexity of participation in the rulemaking by small entities.

"(b) Prior to publication of an initial regulatory flexibility analysis which a covered agency is required to conduct by this chapter—

(1) a covered agency shall notify the Chief Counsel for Advocacy of the Small Business Administration and provide the Chief Counsel with information on the potential impacts of the proposed rule on small entities and the type of small entities that might be affected;

(2) not later than 15 days after the date of receipt of the materials described in paragraph (1), the Chief Counsel shall identify individuals representative of affected small entities for the purpose of obtaining advice and recommendations from those individuals about the potential impacts of the proposed rule;

(3) the agency shall convene a review panel for such rule consisting wholly of full time Federal employees of the office within the agency responsible for carrying out the proposed rule, the Office of Information and Regulatory Affairs within the Office of Management and Budget, and the Chief Counsel;

(4) the panel shall review any material the agency has prepared in connection with this chapter, including any draft proposed rule, collect advice and recommendations of each individual small entity representative identified by the agency after consultation with the Chief Counsel, on issues related to subsections 603(b), paragraphs (3), (4) and (5) and 603(c);

(5) not later than 60 days after the date a covered agency convenes a review panel pursuant to paragraph (3), the review panel shall report on the comments of the small entity representatives and its findings as to issues related to subsections 603(b), paragraphs (3), (4) and (5) and 603(c), provided that such report shall be made public as part of the rulemaking record; and

(6) where appropriate, the agency shall modify the proposed rule, the initial regulatory flexibility analysis or the decision on whether an initial regulatory flexibility analysis is required."[58]

No Small Business Advisory Review (also referred to as "SBREFA Panel") was held for the proposed rule, however. Instead, EPA determined and certified that the rule would "not, if promulgated, have a significant economic impact on a substantial number of small entities." Where such a certification is made, no initial or final regulatory analysis is required, and thus a SBREFA Panel need not be convened.[59]

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[58] 5 U.S.C. § 609(a), (b).

[59] 5 U.S.C. § 605(b): "Sections 603 and 604 of this title shall not apply to any proposed or final rule if the head of the agency certifies that the rule will not, if promulgated, have a significant economic impact on a substantial number of small entities. If the head of the agency makes a certification under the preceding sentence, the agency

HSIA submits that EPA could not lawfully have certified that the proposed rule banning the use of TCE in spot cleaning lacked SISNOSE. EPA has adopted guidance on making the SISNOSE determination:

> "The lower economic impact threshold is particularly important because it is used to screen out rules that generally will not have a significant economic impact and, therefore, can be presumed not to require an IRFA/FRFA (i.e., if all small entities subject to a rule face economic impacts less than the lower threshold, then the rule may be assigned to the Presumed No SISNOSE Category). For this reason the lower economic impact threshold should be set conservatively, at a level that precludes any reasonable possibility that a rule placed in the Presumed No SISNOSE Category might later be found to impose a "significant economic impact on a substantial number of small entities." The upper threshold defines a level of economic impact that would be unquestionably significant for a small entity. In analyzing previous rules, EPA has often defined the lower threshold as compliance costs of 1% of sales and the higher threshold as compliance costs of 3% of sales as shown in the example in Table 2."[60]

The guidance further states that where the number of small entities subject to the rule and experiencing given economic impact is 1,000 or more, regardless of the percentage these constitute of all the small entities subject to the rule that are experiencing given economic impact, the rule will be presumed ineligible for certification.[61]

Spot cleaning is conducted by dry cleaners, virtually all of which are small businesses. The National Cleaners Association (NCA) estimates that there are some 23,550 retail dry cleaning establishments in the United States, having average sales of $250,797 and average profits of $17,809. Industry suppliers report that 60-90% of retail dry cleaners routinely order TCE for use on the spotting board (14,130 – 21,195 small businesses).

During an EO 12866 meeting on October 3, 2016, NCA provided the foregoing and following information. TCE is one of the most used spotting agents. TCE's effectiveness as a spot remover helps cleaners minimize time spent in stain removal and therefore control labor and operational costs. In most small dry cleaning plants the stain removal technician is the highest paid employee. Depending on the operation, labor represents 25-42% (average 30%) of the dry cleaners' costs. Assuming that only twelve garments a day require five additional minutes of stain removal time, this will add one hour a day to the spotter's labor. Assuming the spotter earns just $35,000 per year, one extra hour per day in a 6-day week, with overtime involved, will result in an extra $7,875 in the spotter's gross wages. It will also result in increased utilities due to six additional hours per week of boiler time and plant operation. It will also result in wasted or slowed production in the pressing department as they wait longer for cleaned

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shall publish such certification in the Federal Register at the time of publication of general notice of proposed rulemaking for the rule or at the time of publication of the final rule, along with a statement providing the factual basis for such certification."

[60] Final Guidance for EPA Rulewriters: Regulatory Flexibility Act as amended by the Small Business Regulatory Enforcement Fairness Act, https://www.epa.gov/sites/production/files/2015-06/documents/guidance-regflexact.pdf, Table 2.

[61] *Id.*

garments, further increasing labor costs. Between labor and utilities, NCA estimated *an increased cost of between 4–5% of gross sales.*[62]

Even the lowest increased cost estimated by NCA (4% of gross sales), at the low end of the range of small dry cleaning entities (14,130), constitutes SISNOSE as defined in EPA's guidance. The economic analysis in the docket acknowledges a much larger universe of dry cleaning that use spot removers (48,602) but concludes, with no factual support, that all of these are expected to experience cost impacts that are less than one percent of their revenues.[63]

Remarkably, neither the preamble to the proposed rule nor the economic analysis contains a detailed "statement providing the factual basis for such certification [of no SISNOSE] required by law." Rather, the latter includes a remarkably abstruse discussion of "market failure" that could be inserted into any analysis to support regulation in the absence of data specific to an industry or small business sector.[64] It is respectfully submitted that this does not meet the requirements of the Regulatory Flexibility Act.

III. Failure to Comply with Notice Requirements of TSCA and Administrative Procedure Act

EPA's TCE Work Plan Assessment is legally deficient in a more fundamental way. The draft Assessment was entitled "Degreaser and Arts/Crafts Uses." It states that "EPA focused the assessment on uses of TCE as a degreaser (i.e., both in small commercial settings and by consumers or hobbyists) and on consumer use of TCE in products used by individuals in the arts and crafts field" (p. 14). Spot cleaning is mentioned only in fn. 8: "there were several spot cleaners for fabrics marketed to consumers, but none contained TCE; lists of ingredients were not available for a few of the spot cleaners." There was no reference at all to spot cleaning in the workplace. Yet, with no explanation, the final TCE Work Plan Assessment is entitled "Degreasing, *Spot Cleaning* and Arts & Crafts Uses" and includes "Commercial use of TCE as a spotting agent at dry cleaning facilities" (p. 26).

The failure to notify dry cleaners that EPA was assessing a key agent upon which they rely clearly violates TSCA § 6(b)(4)(H), which states: "The Administrator shall provide no less than 30 days

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[62] https://www.reginfo.gov/public/do/viewEO12866Meeting?viewRule=true&rin=2070-AK03&meetingId=2352&acronym=2070-EPA/OCSPP

[63] *See* Economic Analysis of Proposed TSCA Section 6 Action on Trichloroethylene in Dry Cleaning Spot Removers and Aerosol Degreasers, at ES-15. The difference in number of establishments is due to EPA's reliance on data from decades ago when dry cleaning was a much larger sector.

[64] It begins:

> "Market failure can justify government regulation; the major types of market failures include the following:
>
> - Negative externalities, common property resources, and public goods;
> - Market power;
> - Inadequate or asymmetric information.
>
> The occurrence of any of these conditions justifies further inquiry into the need for government regulation to reduce inefficiencies in the allocation of society's resources. This section describes why negative externalities and inadequate or asymmetric information are present in the market for dry cleaning spot removers and aerosol degreasing products."

Id. at 2-2.

public notice and an opportunity for comment on a draft risk evaluation prior to publishing a final risk evaluation." That this is an "applicable requirement[] of § 6" for purposes of TSCA § 26(l)(4), which sets forth the requirements for EPA to rely upon risk assessments completed prior to enactment of the Lautenberg Act, should be obvious. In addition, § 553 of the Administrative Procedure Act (APA) requires all federal agencies to provide public notice and an opportunity for comment on all proposed rules.[65] The APA definition of "rule" is broad and encompasses background data upon which the rule is based.

Because there was no notice that EPA was addressing spot cleaning, there was no participation by dry cleaner representatives and no peer review of the spot cleaning assessment. EPA based estimates of workers/bystanders on census data "not adjusted to exclude job categories that likely would not be present at dry cleaning facilities. Thus, EPA's estimate likely overestimates the size of the population exposed."[66] Moreover, EPA relied solely on a 2007 California study, which it recognized may not be representative of US dry cleaning facilities. As dry cleaners had no notice that EPA was assessing spot cleaning in the workplace, they did not have an opportunity to comment on the exposure estimates or the study. Thus, the minimal requirements of administrative procedure have not been met in this rulemaking.

An equally serious notice issue is presented by EPA's acknowledgement that it only evaluated the commercial use of TCE for spot cleaning at dry cleaning facilities in the final Work Plan Assessment in response to a peer reviewer comment. It is therefore obvious that the evaluation of this additional use in the final risk assessment was not itself actually peer reviewed. Similarly, the supplemental analyses conducted by EPA to identify risks for the commercial aerosol degreasing use scenario and for various parameters of exposure scenarios for TCE spot cleaner use in dry cleaning facilities were only done long after completion of the Work Plan Assessment and after passage of the Lautenberg Act. Further, these analyses have not been peer reviewed. As noted above, peer review of these analyses is required by the OMB Final Information Quality Bulletin for Peer Review and TSCA.

IV. EPA's Reliance on Alternatives is Unrealistic

TSCA § 6(c)(2) provides:

> "(C) CONSIDERATION OF ALTERNATIVES.—
>
> "Based on the information published under subparagraph (A), in deciding whether to prohibit or restrict in a manner that substantially prevents a specific condition of use of a chemical substance or mixture, and in setting an appropriate transition period for such action, the Administrator shall consider, to the extent practicable, whether technically and economically feasible alternatives that benefit health or the environment, compared to the use so proposed to be prohibited or restricted, will be reasonably available as a substitute when the proposed prohibition or other restriction takes effect."

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[65] 5 U.S.C. § 553(b), (c): "General notice of proposed rulemaking shall be published in the Federal Register, unless persons subject thereto are named and either personally served or otherwise have actual notice thereof in accordance with law. . . . After notice required by this section, the agency shall give interested persons an opportunity to participate in the rulemaking through submission of written data, views, or arguments with or without opportunity for oral presentation."

[66] TCE Work Plan Assessment, at 116.

The proposal suggests that n-propyl bromide (nPB), perchloroethylene, methylene chloride, and water-based compounds could be used as alternatives to TCE in spot cleaning. Many of these alternatives are ineffective, hence the continued market dominance of the TCE-based products. Moreover, there is serious question whether a number of these alternatives would realistically be available, given the designation of nPB, perchloroethylene, and methylene chloride as priorities for risk evaluation/regulation under TSCA § 6(b)(2)(A).[67]

Query how compounds such as nPB could be considered a "reasonably available" substitute for TCE, much less how EPA could consider making such a finding in light of the fact that substitution on nPB in foam fabrication following reduction of the workplace limit for methylene chloride is regarded as a textbook example of "regrettable substitution." Unlike TCE, which has a long history of safe use in the workplace, the serious health impairments suffered by workers in those facilities have been widely documented. Moreover, an nPB industry representative stated at EPA's February 14, 2017 meeting on scoping documents for the ten priority compounds that nPB is no longer used in dry cleaning at all.

V. Gap Filling Purpose of TSCA

As originally enacted and as updated by the Lautenberg Act, TSCA requires EPA to consult and coordinate with other federal agencies "for the purpose of achieving the maximum enforcement of this Act while imposing the least burdens of duplicative requirements on those subject to the Act and for other purposes."[68] Worker and consumer health and safety fall under the jurisdictions, respectively, of OSHA and the Consumer Product Safety Commission (CPSC). The use of TCE in spot cleaning and aerosol degreasing is already more than adequately regulated under the OSH Act and/or the Federal Hazardous Substances Act. This comprehensive regulatory framework provides adequate protections with respect to the same potential adverse impacts and potential exposure pathways targeted by the proposed rule. Taking steps that may lead to the removal of products from the marketplace because workers or consumers failed to comply with the existing legal requirements is not consistent with TSCA either as initially enacted or as revised.

The basis for EPA's broad assertion of jurisdiction over occupational and consumer uses is unclear. The Lautenberg Act eliminated the requirement in TSCA § 6(a) that EPA protect "against [unreasonable] risk using the least burdensome requirements," but did not materially change the existing framework that requires unreasonable risks to be addressed under statutory authority other than TSCA wherever possible. EPA's longstanding interpretation of this framework is as follows:

> "Under section 9(a)(1) of TSCA, the Administrator is required to submit a report to another Federal agency when two determinations are made. The first determination is that the Administrator has reasonable basis to conclude that a chemical substance or mixture presents or will present an unreasonable risk of injury to health or the environment. The second determination is that the unreasonable risk may be prevented or reduced to a sufficient extent by action taken by another Federal agency under a Federal law not administered by EPA. Section 9(a)(1) provides that where the Administrator makes these two determinations, EPA must provide an opportunity to the other Federal agency to assess the risk described in the report, to interpret its own statutory authorities, and to initiate an action under the Federal laws that it administers.
>
> "Accordingly, section 9(a)(1) requires a report requesting the other agency: (1) To determine if the risk may be prevented or reduced to a sufficient extent by action taken

[67] 81 Fed. Reg. 91927 (Dec. 19, 2016).

[68] TSCA § 9(d).

under its authority, and (2) if so, to issue an order declaring whether or not the activities described in the report present the risk described in the report.

"Under section 9(a)(2), EPA is prohibited from taking any action under section 6 or 7 with respect to the risk reported to another Federal agency pending a response to the report from the ether Federal agency. There would be no similar restriction on EPA for any risks associated with a chemical substance or mixture that is not within the section 9(a)(1) determinations and therefore not part of the report submitted by EPA to the other Federal agency."[69]

It was clear from the outset that TSCA is to be used only when other statutes fail to provide a remedy for unreasonable risks. When TSCA was enacted in 1976, Representative James Broyhill of North Carolina indicated that "it was the intent of the conferees that the Toxic Substance Act not be used, when another Act is sufficient to regulate a particular risk."[70] TSCA § 9(a) is substantively unchanged by the Lautenberg Act. The House Energy and Commerce Committee Report states: "H.R. 2576 reinforces TSCA's original purpose of filling gaps in Federal law that otherwise did not protect against the unreasonable risks presented by chemicals," and further clarifies that "while § 5 makes no amendment to TSCA § 9(a), the Committee believes that the Administrator should respect the experience of, and defer to other agencies that have relevant responsibility such as the Department of Labor in cases involving occupational safety."[71]

Colloquies on the floor of the House of Representatives make this intent clear with specific reference to TCE, most notably the following:

"Mr. SHIMKUS. Mr. Speaker, I yield 2 minutes to the gentlewoman from Tennessee (Mrs. *Blackburn*), the vice chair of the full committee.

Mrs. BLACKBURN. Mr. Speaker, I do rise in support of the amendments to H.R. 2576, and I congratulate Chairman *Shimkus* on the wonderful job he has done. Mr. Speaker, I yield to the gentleman from Illinois (Mr. *Shimkus*) for the purpose of a brief colloquy to clarify one important element of the legislation.

Mr. Chairman, it is my understanding that this bill reemphasizes Congress' intent to avoid duplicative regulation through the TSCA law. It does so by carrying over two important EPA constraints in section 9 of the existing law while adding a new, important provision that would be found as new section, 9(b)(2).

It is my understanding that, as a unified whole, this language, old and new, limits the EPA's ability to promulgate a rule under section 6 of TSCA to restrict or eliminate the use of a chemical when the Agency either already regulates that chemical through a different statute under its own control and that authority sufficiently protects against a risk of injury to human health or the environment, or a different agency already regulates that chemical in a manner that also sufficiently protects against the risk identified by EPA.

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[69] 4,4'-Methylenedianiline; Decision to Report to the Occupational Safety and Health Administration, 50 Fed. Reg. 27674 (July 5, 1985). EPA also has acted under § 9(a) to refer 1,3-butadiene and glycol ethers to OSHA, 50 Fed. Reg. 41393 (Oct. 10, 1985) and 51 Fed. Reg. 18488 (May 20, 1986), respectively, and to refer dioxins in bleached wood pulp and paper products to the Food and Drug Administration, 55 Fed. Reg. 53047 (Dec. 26, 1990).

[70] 122 Cong. Rec. H11344 (Sept. 28, 1976).

[71] H. Rep. No. 114-176 (114th Cong., 1st Sess.) at 28.

Would the chairman please confirm my understanding of section 9?

Mr. SHIMKUS. Will the gentlewoman yield?

Mrs. BLACKBURN. I yield to the gentleman from Illinois.

Mr. SHIMKUS. The gentlewoman is correct in her understanding.

Mrs. BLACKBURN. I thank the chairman. The changes you have worked hard to preserve in this negotiated bill are important. As the EPA's early-stage efforts to regulate methylene chloride and TCE under TSCA statute section 6 illustrate, they are also timely.

EPA simply has to account for why a new regulation for methylene chloride and TCE under TSCA is necessary since its own existing regulatory framework already appropriately addresses risk to human health. New section 9(b)(2) will force the Agency to do just that.

I thank the chairman for his good work."[72]

Indeed, TSCA § 9 was strengthened by the Frank R. Lautenberg Chemical Safety for the 21st Century Act, and it was clear from the outset that TSCA is to be used only when other statutes fail to provide a remedy for unreasonable risks. Representative James Broyhill of North Carolina indicated that "it was the intent of the conferees that the Toxic Substance Act not be used, when another act is sufficient to regulate a particular risk."[73] EPA applied this statutory directive in determining that the risk from 4,4' methylenedianiline (MDA) could be prevented or reduced to a significant extent under the Occupational Safety and Health Act, and referring the matter for action by OSHA.[74] And in an analysis of TSCA § 9, EPA's Acting General Counsel concluded that "Congress expected EPA – particularly where the Occupational Safety and Health Act was concerned – to err on the side of making referrals rather than withholding them."[75]

There is no evidence that EPA has submitted to OSHA "a report which describes such risk and includes in such description a specification of the activity or combination of activities which the Administrator has reason to believe so presents such risk and includes in such description a specification of the activity or combination of activities which the Administrator has reason to believe so presents such risk," as required by TSCA § 9(a)(1). The non-existent report obviously did not "include a detailed statement of the information on which it is based" and was not "published in the Federal Register," as required.

Had the required report been issued, it presumably would have identified how OSHA's authority over the workplace was insufficient to address the risks posed by spot cleaning and aerosol degreasing using TCE. A letter from the Assistant Secretary of Labor for Occupational Safety and Health (undated but apparently issued on April 4, 2016) identifies limits on OSHA's authority to regulate hazardous substances such as TCE, but it does not come close to meeting the requirements of TSCA for EPA action in this case. The April 2016 letter identifies no gap specific to spot cleaning or aerosol degreasing in any particular category of workplace, rather it simply recites how OSHA's authority does not extend to self-employed

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[72] 162 Cong. Rec. H3028 (May 24, 2016).

[73] 122 Cong. Rec. H11344 (Sept. 28, 1976).

[74] 50 Fed. Reg. 27674 (July 5, 1985).

[75] Memorandum to Lee M. Thomas from Gerald H. Yamada, June 7, 1985, p. 2.

workers, military personnel, and consumer uses. But those are limitations that were imposed by Congress and have existed since the Occupational Safety and Health Act was enacted (six years before enactment of TSCA). Those limitations apply to every use of every toxic substance. Congress cannot have meant, in enacting "gap-filling" legislation, to open the door to EPA assuming all authority over the use of hazardous substances in the workplace.

If EPA were to identify a category of exposure deemed to present a risk that is unreasonable, these considerations indicate that referral under § 9(a) would be the appropriate course.[76] It is clear from Section 9(a) that TSCA is to be used only when other statutes fail to provide a remedy for unreasonable risks.

Attachments:

Appendix 1
Appendix 2
Appendix 3
Appendix 4
Appendix 5

[76] As noted above, TSCA § 9(a) provides that if the Administrator has reasonable basis to conclude that an unreasonable risk of injury is presented, and he determines, in his discretion, that the risk may be prevented or sufficiently reduced by action under another federal statute not administered by EPA, then the Administrator shall submit a report to that agency describing the risk. In the report, the Administrator shall request that the agency determine if the risk can be prevented or sufficiently reduced by action under the law administered by that agency; if so, the other agency is to issue an order declaring whether the risk described in the Administrator's report is presented, and is to respond to the Administrator regarding its prevention or reduction. The Administrator may set a time (of not less than 90 days) within which the response is to be made. The other agency must publish its response in the Federal Register. If the other agency decides that the risk described is not presented, or within 90 days of publication in the Federal Register initiates action to protect against the risk, EPA may not take any action under § 6 of TSCA.